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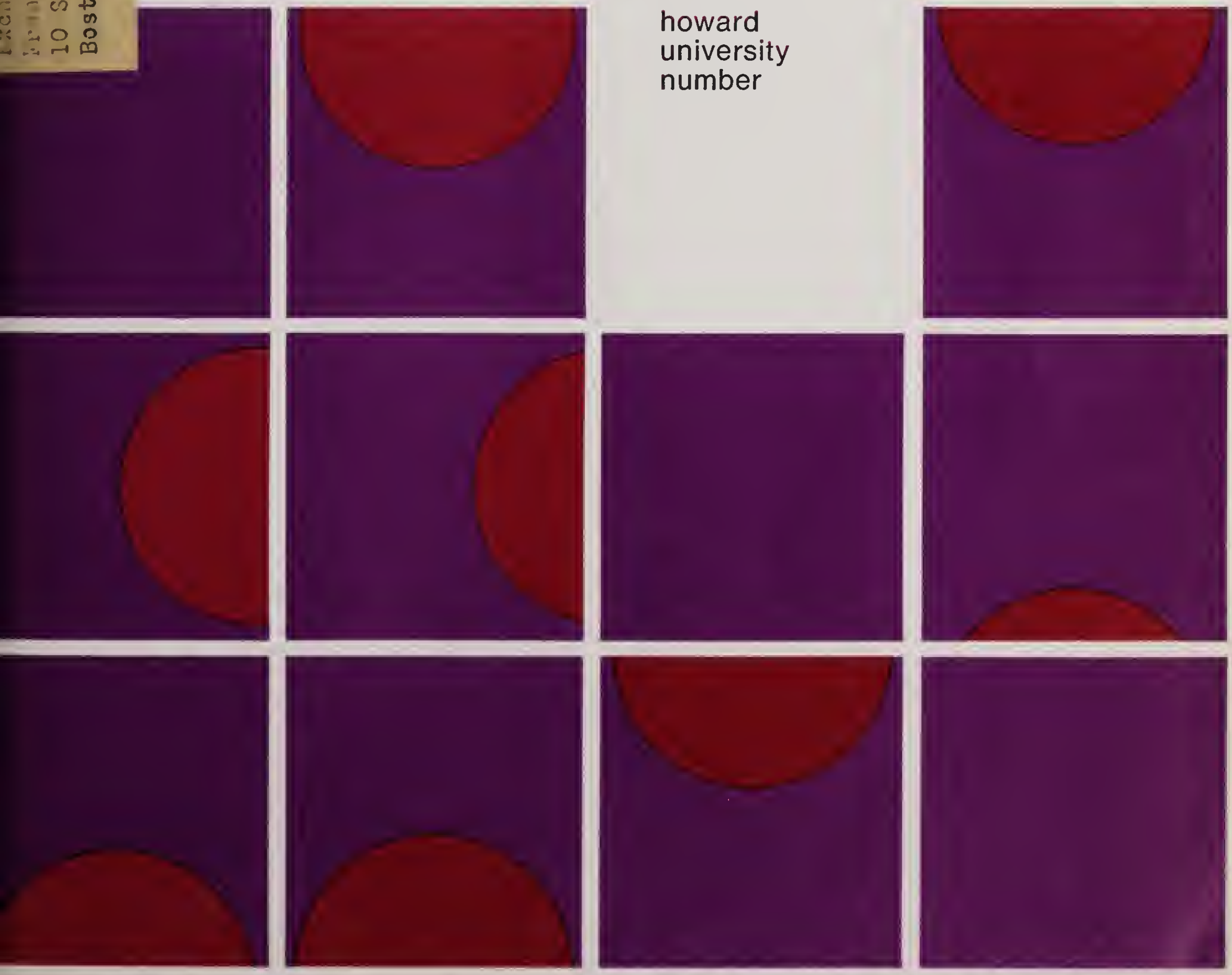

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PRESIDENT'S MESSAGE

Ring out, wild bells, to the wild sky,
The flying cloud, the frosty light:
The year is dying in the night;
Ring out, wild bells and let him die.

Tennyson

HAPPY NEW YEAR!

Darrell C. Crain, M.D.

The "new" Annals which greets you today is symbolic as well as specific evidence of innovations planned by your officers, board members and committee chairmen for 1972. Combining the scientific excellence of Wallace Yater, Editor, with the imaginative creativity of Raymond Scalettar, Associate Editor, and the technical brilliance of Judith Perruso, Assistant Editor, the MEDICAL ANNALS OF THE DISTRICT OF COLUMBIA is destined to be one of the great State journals of our country.

The Annual Scientific Assembly, too, will mark a complete change. Most important is the unusual program being completed under the direction of Margaret Kenrick. Six in depth "Seminar Work-Shops" concurrently in session two hours each morning of the three day convention, will replace the usual series of unrelated lectures. Registrants will thus have six hours to consider such topics as "Sex, Venereal Disease and Abortions," " Heroin, Marijuana, LSD and Alcohol," "Surgical Controversies" and others. For the first time in the history of our Society the Assembly will be held outside of Washington. The Greenbrier - in White Sulphur Springs, West Virginia - has been described by William Kurstin, General Chairman, as "the most beautiful resort hotel in the United States, with unsurpassed convention facilities." Full details of this outstanding event will be forthcoming in the announcements by Lewis Kurtz and his Publicity Committee - including news of the golf tournament and other events being planned by Alvin Kay's Sports Committee.

Changes of great significance in the Society's committee structure have also been instituted. Lomax Wells is chairing a new committee on "Provisions of Health Care" to investigate all facets of how people in our city obtain medical care, including comparative studies of hospital practice, conventional fee for service practice, and pre-paid group practice. Elbert Phelps' "Hospital and In-Patient Care" Committee will study and make recommendations on extended care facilities and nursing homes. The Fee Review Committee, under Howard Ticktin, will analyze current partitions of the Blue Cross-Blue Shield dollar.

Your new Annals will carry reports of these and numerous other committee activities which are of vital interest to every member of our Society.

But January is named for Janus, the Roman god with two faces, and that which looks back is equal to that which looks forward, so we shall not fail to recognize and continue the outstanding programs initiated by President Marvin Korengold and ably implemented by Executive Director Franc Ferraraccio, in the epic year of 1971. The Drug Dependency Committee under Donald Glew will continue to involve our Society in this vital facet of urban and suburban life; peer reviews will continue to expand under Edwin Parker's Utilization Committee, Wesley Oler's Grievance Committee, and Ernest Gould's Ethics Committee. The Society Newsletter, so auspiciously started under President-Elect Burka will thrive under the same committee, now to be headed by James Kehoe. And the Society dinners - which have unified our membership as no other factor - will be continued (with open bar). The first is scheduled for an evening in March at the Sheraton Park Hotel.

So it will be an active year. And we shall bear in mind that our Society will thrive, and prosper, and serve the interests of our members as it best serves the health needs of our community.

As President of the Medical Society of the District of Columbia, I shall need the support of every member to accomplish these important goals. I know I can count on you.

Darrell C. Crain

THE MEDICAL ANNALS . . .

With this issue of the MEDICAL ANNALS OF THE DISTRICT OF COLUMBIA we hope to bridge the gap between the traditions of the past and the realities of today and the future. We want our Journal to continue to reflect significant aspects of medical science, and be a meaningful chronicle of what our medical profession is doing and thinking.

Today, as our profession is under attack, as Congress debates new health legislation and as the health delivery system in this country comes under greater scrutiny, we must tell our story clearly and forthrightly. A change in journal cover and format alone will not do this. Instead we need greater involvement and input from our membership not only in the field of scientific articles, but with regard to the medical aspects of our complex society.

A new Editorial Board has been selected and we hope there will be a presentation of cogent and timely ideas. These opinions will be those of the individuals who write the editorials and not the official policy of the Medical Society of the District of Columbia. Some of these ideas will be controversial. To those who disagree or wish to make comment, we invite the opportunity to reply via a letter to the editor.

Some of the changes that are in the progress of development will be noted in this issue. A Medico-Legal column will be the responsibility of Mr. Stefan Tucker, a distinguished Washington attorney. A Continuing Medical Education column will be provided by the Continuing Medical Education Committee under the chairmanship of Dr. Jay Shapiro. Relevant special articles will appear regularly. Summaries of important scientific meetings held at the Medical Society will be forthcoming. A Question of the Month column will subsequently appear under the auspices of Dr. William Jaffurs.

These and other innovations are planned. To succeed, we will need the help of our membership and subscribers. We know we cannot please everyone with these changes and those that are forthcoming, but in the process of change we will make our Journal more relevant.

There are many devoted physicians who spend many hours in Committee Meetings at our Medical Society. The opinions and recommendations of these Committees encompass the collective wisdom of many of the physicians of our community. These ideas must be brought to life and communicated to our membership and the profession of this metropolitan area. This is a dynamic Medical Society whose numerous accomplishments of this past year were enumerated by our past president, Dr. Marvin Korengold, at our annual meeting of November 15, 1971. We hope to make our Journal even more dynamic and meaningful, while steadily increasing its scientific merit.

Raymond Scalettar, M.D., F.A.C.P.
Associate Editor

THE COST OF MEDICAL CARE

The cost of medical care is one of the urgent domestic issues in the United States today - not only for the medical profession but for all Americans. Politicians, it seems, are particularly concerned with the cost of health care.

As physicians, we are caught somewhere in the middle. The public cannot separate hospital charges from physician fees. It is incumbent upon the medical profession to help restrain rising hospital costs. Failure to achieve this will almost certainly result in government regulation.

Utilization committees are hard at work in our medical society, and in other societies throughout the country, in an attempt to curb these rising costs. One aspect of utilization seems obvious and would be an economy measure available to all physicians regardless of specialty. Imagine the savings realized if only one laboratory examination was omitted for each hospitalized patient! Even in these days of "defensive" medicine, when physicians are acutely aware of ordering complete laboratory studies, there is a need for greater discrimination in this regard.

Those of us who serve on the staff of teaching institutions must be especially cognizant of this aspect of medical care. Excessive and often repetitious laboratory studies are no more excusable when ordered by house staff or medical students than by the attending physician. Furthermore, indiscriminate laboratory examinations are anathema to medical education as well as to quality patient care.

Jerome W. Canter, M.D., F.A.C.S.

ULCER, ANYONE...?

In 1911 Dr. Bertram W. Sippy - of Sippy diet fame - enlightened the medical community with his new and "as radical as I dare publish" treatment for peptic ulcer (1). Four years later having obtained "further results beyond belief" he could then expound uninhibitedly on his now modified regimen to remove gastric juice corrosion and promote ulcer healing (2). "The uncomfortable preliminary starvation period serves no purpose when the acidity is controlled and has, therefore, been entirely abandoned..." he wrote. Four weeks of incessantly administered heavy calcined magnesia with sparingly allowed bland items mouthfuls rarely failed. Dr. Sippy struck a hard slap onto the hands of surgeons with his uniformly successful medical program to include high grade gastric outlet obstruction.

Over the last sixty years, in spite of continued censure, nothing has remained so solidly undisturbed in the practice of medicine as the Dr. Sippy inside everyone of us. Heavy calcined magnesia has developed into a multimillion dollar antacid industry of wide selection in all but data to support its worth beyond their analgesic effect (3,4). Antacids relieve pain, indeed they do; whether they speed up healing of an ulcer or prevent its recurrence is entirely another matter. The available information suggests they don't, but no serious effort is in sight to answer the question - which instead seems to be, not whether they work, but which one works better! Hardly any arguments can be mustered for the long term value of anticholinergic medication. Nevertheless they are offered reflexly, along with the antacid of choice, with hopeful expectations of therapeutic symbiosis. Under increasingly heavy attack, diet remains alive but ill stricken and slowly deteriorating (5,6,7,8).

Understandably, then, from the mainstream of believers a few highly seasoned iconoclasts have detached themselves into an agnostic group that forms the other side of the controversy in the therapy of peptic ulcer (9,10,11). Not only for antacids, anticholinergics or diet but for all the traditional interdictions constantly befalling the ulcer patient. So today, 3 scores later - with the majority undecided, with great physiological sophistication, with obsessive insistance on controls and irrational love for P values, - we still heatedly express our diverging views on the salutary effects of psychotherapy, liquorice, bed rest, smoking, the New Year's Eve use of dilute alcohol or even a sporadic sneak to the Mexican carry out. And to confuse the issue irreparably most patients do well regardless of physician prejudice or therapeutic enterprise.

But then came melena...twice...and the nerves were cut, and it felt so good thereafter. And the surgeons slapped back with equal vigor - "results beyond belief to you too, sir..." Incredible. Sixty years to go back where we started? Not really, but it almost looks that way. Painfully we must admit ignorance into the crucial questions of the ulcer story - a vital issue to an underestimated (12) 10% of the population. Therapeutic complacency has thwarted progress; etiology and pathogenesis have gone begging. The time honored dictum "no acid - no ulcer" has fallen (13); hopefully the sting will provoke some thoughtful reflections...which reminds me - why so many ulcers in the bulb and not in the fundus where the juice runneth over? Why so much relief of pain with one dose of antacid when the pH raises above 3.5 for only a few minutes? Why the spring and fall relapses? The blood group relation? The classic symptoms without the crater? The invariable healing of a full thickness gastric mucosal biopsy exposed to as much acid as the ulcer located 1 cm. away? Why do one-third to one-half of asymptomatic executives have a duodenal ulcer and never know about it - in other words the genesis of symptoms? What about the "stress" of a "stress ulcer" versus the "tension" of a "regular ulcer?" The influence of catecholamines, hormones or drugs on ulcer provocation? Or even why do ulcer patients form more than three vertical furrows on the frowned forehead(14)? These are only a few of the many interrogations for which there are no answers. I suppose if we don't get busy in the next sixty years we will have sixty new antacids and still no answers.

Rafael A. Rivera, M.D.

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THE COTTAGE PHYSICIAN

Every so often, the editorialist is favored with a simple, spontaneous and moving demonstration that brightens his day and lightens his burden. A colleague recently received the following letter:

> *"...It was a great experience to us this last Saturday about 5:30 p.m. My wife was not feeling well and my son and daughter-in-law suggested we call you.*
>
> *We called and in about 3 to 5 minutes a very kind and patient person returned our call and was very understanding and explained the possibilities of any symptoms and asked to speak to me and I certainly felt much relieved by his patient explanation.*
>
> *I really want to thank him for his thoughtfulness and tell you that he is one human that you are fortunate to have as a stand-by or whatever his capacity is. I would have sent him a separate note but I would think you and he should be complimented. Much thanks..."*

Now, how does this very kind note raise a question worthy of editorial comment? Well, a great deal is being written, debated, argued, and sometimes shouted about the need for the development of new methods of "delivering" medical care. Unfortunately, most of this debate generates more heat than light. Proponents of new plans are often guilty of wishfully thinking that major changes from the present system (or "non-system") are automatically changes for the better. Similarly, those who would defend the present state of affairs sometimes appear to be unreasonably defensive regarding the mere possibility of change. The obvious defect in this noisy debate is simply the appalling dearth of hard facts. Fortunately, it now seems that belated attempts are being made to get at these facts--i.e., are HMO's more efficient at providing medical service than the private physician functioning in his "cottage industry" office? Can government run an insurance program more efficiently than private companies have done? And on and on.

But where, oh where, in the statistical surveys and economic charts do we plug in the value to the public or the service alluded to in the letter printed above? How does the cost analyst place a value on that phone consultation? This service, in this case to an absolute stranger, probably occurs thousands of times for each thank-you note actually written. It was a service performed free of charge and with no need for the backing of a complex clinic or a fully-staffed emergency room or complicated table of organization. It simply required dedication, empathy and grace. These are commodities that are hard to come by. If they are lost because of hastily contrived change, they may never be retrieved. Caveat Emptor.

Paul Schlein, M.D.

PEER REVIEW, QUALITY CONTROL, AND INSPECTIONS

The AMA definition of peer review is "evaluation by practicing physicians of the quality and efficiency of services ordered or performed by other practicing physicians. Peer review is the all inclusive term for medical review efforts."

Most of the practical application of peer review has been to the economy of practice. A peer review system without economic implications exists, however, in the Air Force, and the contrast between peer scrutiny as compared to the structured and sometimes rigid inspection systems is perhaps instructive.

The oldest Air Force quality control mechanism operates under its Inspector General, as in the other military services. The inspections cover every unit in the Air Force, with the medical function modified to evaluate medical care systems chiefly from a management standpoint. Its several physician members are usually senior and able administrators, but are rarely patient-care oriented at this stage of their careers and report directly to non-medical authority and not to the Surgeon General. Other formal inspection systems are much the same, operating from subordinate commands. These teams are expected to provide guidance and assistance as well as to log discrepancies.

Complementing the military system are the American Hospital Association review of Air Force Hospitals, teaching hospital reviews by the appropriate professional authorities, the usual review committees within the hospitals themselves, and various other reviews in individual situations. These inspecting agencies are concerned with the total medical unit, although an individual may stand out if he departs far enough from the norm (such as by performance of unjustified surgery).

Since the hospital's function of medical care is not always reflected in committee reports, budget statements, and other matters of administrative importance, a system of peer review evolved in the Air Force. Although at first intended to provide consultation, lectures, and rounds, the Consultant Program developed into a good teaching and communication device and an excellent professional review technique. The half dozen major hospitals send out teams of senior specialists to their professional counterparts at smaller referral hospitals, who in turn send teams to the smaller community-level Air Force hospitals to round, lecture, and inevitably review and observe. The process is usually enjoyable on both sides, as professional communication tends to be, and tactful guidance is surprisingly easy (usually anyway). The consultants report to the Surgeon General are almost always favorable (except for criticism of areas where budgets are critical). At the working level of the consultant and his colleagues there is little friction, but the anguish of the hospital commander is directly proportional to his removal from practice and direct concern with patient care as when a hospital commander, whose only contact with the concept of cardiac monitoring has been to ignore it, is written up for not having it.

What does this mean to "the private sector"? The inspector systems are generally good administratively, but IG reports have hurt good physicians whose administrative failings, often minor, have been more obvious than superior professional virtues. Too, IG reports are often more concerned with the absence of signatures from records than the content of the record. The Consultant Program, on the other hand, is concerned with the medical side of patient care, deals with individual physicians, provides the specialist, who alone is competent to evaluate his colleagues' practice of their specialty, and is able to correct mistakes more through teaching than by bureaucratic fiat. Since government systems would more probably follow the simpler bureaucratic model, this amounts to another sounding of the too familiar alarm that we must police ourselves before we are policed (which is true, nonetheless).

In summary, a peer review system has been demonstrated in the Air Force to be practical and even pleasant (at least for that vast majority who are not found wanting). In ten years it has produced a minimum of abrasion while yielding excellent evaluation of working physician performance by other working physicians, in a very large and diverse community. This is very reassuring to know.

Robert B.W. Smith, M.D., F.A.C.P.

scientific articles

Eosinophilia and Metastatic Carcinoma*

MICHAEL V. VIOLA, M.D.†, EDB. CHUNG, M.D., PH.D.‡,
AND MADAN G. MUKHOPADHYAY, M.D.§

The occurrence of increased numbers of eosinophils in the peripheral blood of patients with chronic myelogenous leukemia and Hodgkin's disease is well known.[6,24] Eosinophilia in association with nonhematological malignancies has not received attention in the recent literature, however. In this report we describe a patient with metastatic carcinoma from an unknown site with persistent marked eosinophilia. Well-documented cases of this association in the literature will be reviewed also.

Report of Case

The patient, a 72-year-old black man, was admitted to the hospital on March 4, 1971 because of enlarged cervical lymph nodes of 1 month's duration. He had a chronic nonproductive cough without chest pain, dyspnea, hemoptysis, or weight loss. He had smoked 1 pack a day for 50 years. There was no history of allergies, asthma, or skin diseases. He had been taking no medication and had never traveled outside of the United States.

Physical examination revealed a thin but well-nourished man in no distress. T-98.6, BP-110/80, HR-80 reg, RR-20. There were multiple, enlarged, bilateral cervical, supraclavicular, and axillary lymph nodes. They were hard and non-tender, and the largest measured 4 cm in diameter. The A-P diameter of the chest was increased, and the breath sounds were distant with a few scattered rhonchi. Otherwise the examination was normal.

Laboratory data. Hb. 17 gms%, hct 52%, WBC 16,100, with polys 27, lymphs 18, monos 3, bas 2, eos 50. During the entire hospitalization the WBC ranged from 11,000-16,000 with eosinophil counts from 50-52%. Urinalysis was normal. Stools were negative for ova and parasites. A bone-marrow examination was refused.

BUN 20, glucose 90, Na–141, K–5.0, Cl–100, CO_2–28, uric acid–8.2, total bilirubin–0.3, LDH–225, SGOT–40, SGPT–120, alk phos-50, LDH-240, acid phos–0.3, amylase 111, lipase 1.3, calcium–10.5, phosphorus–3.0, STS–weakly positive, VIT B12–0.018 mcg%, plasma cortisol–20 mcg%. Serum protein electrophoresis–total protein–9.4 gm%, alpha 1–2.8%, alpha 2–9%, beta–9%, gamma–45.3%. The electrophoretic pattern showed a diffuse increase in gamma globulin. Qualitative immunoelectrophoresis– IGG 2,080 mg%, IGA 320 mg%, IGM 360 mg%. Blood gasses– ph 7.42, pO_2 66, p CO_2 36, %sat. 92.4%. EGG– incomplete right-bundle-branch block.

A chest X-ray film showed interstitial fibrosis and a right paratracheal mass. Intravenous pyelogram, thyroid scan, and brain scan were normal. Liver scan was strongly suggestive of metastases.

Sections of the lymph node taken from the left external jugular chain showed that the lymphoid tissue was largely replaced by sheets and clusters of malignant epithelial cells. These cells were round to polygonal, and their cytoplasm was pale pink, granular, and often vacuolated. The nucleoli were large and pleomorphic, and often contained prominent nucleoli and clumped chromatin. Occasionally bizarre multinucleated forms and atypical mitotic figures were noted. In areas the tumor-cell clusters were centrally necrotic. The intervening stroma was scanty and often focally desmoplastic. In the vicinity of the tumor necrosis there were scattered, numerous eosinophils along with some plasma cells and lymphocytes (Figs. 1 and 2).

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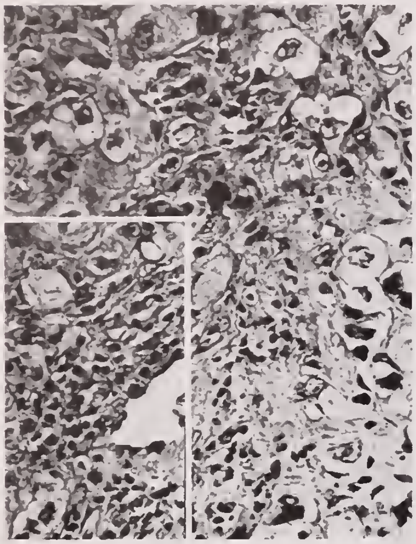

FIG. 1. Metastatic anaplastic carcinoma involving a cervical lymph node. Note neoplastic epithelial cells at lower and left half of photo-micrograph. The majority (approximately 47%) of inflammatory cells around the sinusoid represent eosinophils.

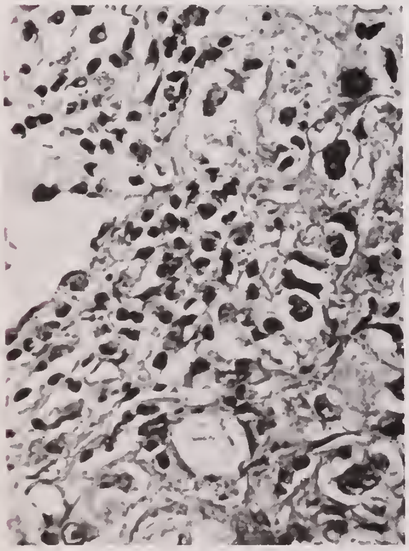

FIG. 2. Higher magnification of inset marked in Fig. 1 showing eosinophilic granulocytes. Twenty-seven eosinophils may be counted.

Discussion

In view of this patient's long smoking history and the paratracheal mass noted on chest X-ray films it is most likely that he had a bronchogenic carcinoma with lymph-node and liver metastases. Bronchoscopy was unsuccessful in demonstrating a lesion, and sputum cytology was negative; Thus a definitive diagnosis of the primary site could not be made.

The common causes of eosinophilia (allergies, skin disease, parasitic infestations, hyper-eosinophilic syndrome, drugs) were not evident. It is most likely that the eosinophilia was provoked by the carcinoma. Supporting this is the heavy infiltration of eosinophils in the tumor specimen. Another explanation for both the mild polycythemia and the eosinophilia is the hypoxemia secondary to chronic obstructive lung disease. Hypoxia can induce eosinophilia in experimental animals[23] and in patients with chronic mountain sickness.[12] However, we have not observed eosinophilia in other patients with chronic obstructive lung disease with prolonged hypoxia. Cigarette smoking has been reported to induce eosinophilia.[22] We have not ruled out tobacco hypersensitivity; the patient refused a short trial of abstinence from cigarette smoking.

Eosinophilia has been reported in association with a number of different carcinomas and sarcomas (table 1). It is more frequently seen with the commonly occurring tumors. Acinar-cell carcinoma of the pancreas has been described recently in association with skin lesions, arthralgias, mild eosinophilia, and high levels of serum lipase.[19] It is thought that fat necrosis in skin and periarticular fat causes the clinical findings as well as the mild eosinophilia.

In cases of eosinophilia with tumors there is often a total leukocytosis, usually below 25,000. However, a number of patients had white blood cell counts in excess of 50,000 and were initially thought to have eosinophilic leukemia. The majority of patients have had normal red blood cell and platelet counts, although some have had a mild anemia. Eosinophilic infiltration of the tumor specimen has been noted in suprisingly few of the reported cases. Necrosis of the tumor has been frequently seen, but considering the frequency of necrotic areas in many tumor specimens this finding is difficult to evaluate.

Issaacson and Rapoport[8] have emphasized that when eosinophilia occurs with malignant diseases it indicates widespread metastases. This is a consistent finding in practically all of the reported cases. Most patients described had metastatic lesions in the liver or abdominal lymph nodes. A number of patients had normal eosinophil counts when the disease was localized, but as the disease disseminated the eosinophil counts increased. Those few patients reported with localized disease had disappearance of the eosinophilia when the tumor was removed. The patient described in this report fits the typical description of widely disseminated neoplasm with areas of necrosis in the tumor.

Numerous hypotheses have been proposed to explain the eosinophilia associated with metastatic carcinomas. 1. Bone-marrow metastases with resultant stimulation of eosinophil release from the marrow was suggested in early reports but bone-marrow metastases have not been documented in any cases reviewed. 2. An increased local mast-cell reaction around the tumor with breakdown of these cells and release of histamine has been suggested as a cause of eosinophilia,[21] but this reaction has never been demonstrated histologically. 3. Elaboration of humoral substances by the tumor (e.g., histamine) which stimulate eosinophil release from the bone marrow has been suggested also. Prolonged infusion of histamine in animals can provoke peripheral blood eosinophilia.[1] Histaminuria has been

TABLE 1: Reported Cases of Eosinophilia with Non-hematological Neoplasms

				(Site of Metastases)			
Type	Number	Tumor Necrosis	Tumor Eosinophilia	Metastases	Liver	Abd.	Bone
Pancreas	9	3		9	8	9	1
Colon	8	3	2	7	3	5	0
Lung	5	2		5	2	1	1
Stomach	5	1		3	2	2	0
Ca (? primary)	4	2	1	4	3	1	0
Cervix	3	0	1	2	1	2	0
Breast	2	0		2	0	1	0
Thyroid	2	0		1	1	0	0
Penis	2	0		0	0	0	0
Sarcoma	2	0		2	1	2	0
Biliary	2	0		2	2	2	0
Adrenal	1	1	1	1	1	1	1
Liver	1	0		0	0	0	0
Total	46	12	5	38	24	26	3

* References 2, 3, 4, 5, 7, 8, 9, 11, 13, 14, 16, 18, 19, 21, 25. Included are cases from foreign literature before 1946 reviewed by Issaacson and Rapoport[19]. Seventeen additional cases were reported but were poorly documented.

detected in certain carcinoid tumors and systemic mastocytosis but not in patients with other carcinomas.[15,17] Also, most experimental tumors have low concentrations of histamine.[20] 4. Eosinophils are intimately involved in immune reactions and one of the functions of this cell is the ingestion of antigen-antibody complexes.[10] The exact specificities of the immune reactants which can stimulate an eosinophilic response are not known. It is possible that tumor-specific antigens or necrotic antigens associated with this tumor provoked an antibody response and formed antigen-antibody complexes of the right specificity to stimulate an eosinophilic response. It is interesting in this light that the patient had a marked hypergammaglobulinemia.

Previous attempts to induce eosinophilia in experimental animals with the use of inocula of the patient's tumor extracts or sera have been unsuccessful.[25] We inoculated our patient's sera into a number of experimental animal species with no resultant eosinophilia.

Summary

A patient is described with metastatic carcinoma to lymph nodes and probably liver from an unknown site with persistent marked peripheral blood eosinophilia. The tumor had an eosinophic infiltrate and areas of necrosis. A review of the literature indicates that eosinophilia may occur with any type of carcinoma. The association is most frequent in the presence of widespread metastatic disease, particularly to the liver and abdominal lymph nodes. Possible mechanisms of eosinophilia in metastatic carcinoma have been discussed.

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Corynebacterium Vaginale*
An Analysis of 68 Isolations

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In 1953 Leopold[1] isolated a small gram-negative aerobic bacillus from the urine of males and from patients with cervicitis. This organism was later named *Hemophilus hemolyticus vaginalis* by Lutz et al.[2] Gardner and Dukes[3] described a similar organism under the name of *Hemophilus vaginalis* in patients with nonspecific vaginitis. The organism was subsequently reclassified and is presently known as *Corynebacterium vaginale.*[4]

Corynebacterium vaginale has been isolated with increased frequency at Freedmen's Hospital, and we were interested in studying its disease–causing potential in a hospital population composed predominantly of Negro patients.

Materials and Methods

All vaginal and cervical swabs submitted for culture were reviewed during the period 1966 through 1969. Each specimen was inoculated into trypticase soy broth with 5% sheep or human blood and thioglycolate medium, and streaked onto blood agar plates. These were incubated aerobically at 37°C for 48 hours. Various carbohydrates were used for aerobic fermentations, and in-vitro sensitivity studies were performed by the use of commercial antibiotic discs.

Results

A total of 413 cultures were performed during the period of the study and *C. vaginale* was isolated in pure and mixed cultures from 68 patients (Table 1). The results of the sensitivity studies are noted in Table 2.

TABLE 1. Sources and Distribution of Cultures of *C. vaginale*

	Cultures		
Source	Pure	Mixed	Total
Vaginal Secretions	37	20	57
Lochia	6	5	11
Total	43	25	68

Case Studies

Each case was studied with emphasis on the association of clinical symptoms and signs with the isolation of *C. vaginale* in pure culture and the disappearance of symptoms following specific antibiotic therapy.

The patients ranged in age from 18 to 56; the majority were in the child-bearing age and came in with vaginal discharge or leucorrhea. Some complained of pruritus, but the majority did not mention this as a problem. Cultures of lochia were submitted because of changes in the color of the discharge.

Of the 37 patients having pure cultures of the organism, only 3 failed to respond to the initial course of specific antibiotic therapy. These patients were re-cultured, and the same organism was isolated. Following additional antibiotic therapy they responded favorably. Four of the patients with mixed cultures failed to respond initially but responded after further therapy.

There was no follow-up after the patients were discharged from the hospital.

Bacteriology

Cornyebacterium vaginale is a small gram-variable, sometimes gram-negative, tangled, beading bacillus with a diphtheroid arrangement. It grows well on blood agar and trypticase soy agar containing 5% sheep or human blood. It does not require the "X" factor. We did not, however, observe its growth aerobically without the use of blood. Forty-eight hours of incubation, at 37°C, are required for growth. Greater recovery is noted when CO_2 is used.

On blood agar plates, the colonies are minute, colorless, and pinpoint in size. They are surrounded by a clear zone of hemolysis. When enriched

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TABLE 2. Antibiotic Sensitivity Reactions to *C. Vaginale*

Antibiotic	Reaction	Antibiotic	Reaction
Penicillin (10 u)	S	Gantrisin (2 mg)	R
Colymycin	R	Kantrex (30 u)	S
Nalidixic Acid (30 mg)	R	Keflin (30 u)	S
Neomycin (5 ug)	R	Lincocin (2 ug)	S
Bacitracin (10 u)	S	Mandelamine (3 mg)	S
Chloromycetin (30 ug)	S	Polycillin (10 ug)	S
Colistin (10 ug)	R	Streptomycin (10 ug)	S
Erythromycin (15 ug)	S	Tetracycline (30 u)	S
Furadantin (100 ug)	S	Hufcillin (1 u)	S

S = Sensitive R = Resistent

thioglycolate medium is used the upper portion of the medium is routinely heavily inoculated with the organism and then overlaid with 1 ml of ascitic fluid. Within 48 hours the organism grows as small, white "puff-balls" in the upper phase of the medium.

The organism is non-motile, nonencapsulated, oxidase-negative, and devoid of endospores. It is catalase-negative, and nitrate reduction is variable. Growth is inhibited by alpha hemolytic streptococci and pneumococci.

Biochemical reactions are somewhat variable. Indole is not produced. At times, acid is formed from glucose, maltose, dextrin, starch, and glycogen. There are no reactions in lactose, galactose, sucrose, mannitol, and salicin.

Corynebacterium vaginale is sensitive to the majority of antibiotics, but some strains are resistant to penicillin (Table 2).

Discussion

Edmunds[5] found that *C. vaginale* was associated with puerperal fever, trichomonas vaginitis, yeast infections, and the isolation of PPLO. He believes that the organism plays a part in vaginal and cervical pathology where no other pathogen is demonstrated.

In the cases studied at Freedmen's Hospital there appeared to be a definite correlation between the patients' signs and symptoms, isolation of the organism in pure and mixed cultures, and the disappearance of symptoms following specific antibiotic therapy.

If cervical or vaginal biopsies were performed prior to and after therapy, the presence and absence of nonspecific inflammation in the histological sections would prove conclusively the pathogenicity of *C. vaginale*.

In random cultures we noted that *C. vaginale* is not of the normal vaginal flora, and when found is probably pathogenic, whether in pure or mixed culture. These findings have been noted by others.

Summary

An analysis of 68 cases of probable *Corynebacterium vaginale* infection has been presented. The bacteriology and biochemical and sensitivity reactions have been detailed. The organism is probably pathogenic and is destroyed by specific antibiotic therapy.

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Pneumonia and the Alcoholic: An Epidemiologic Study

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It is widely accepted clinical impression that alcoholics are more susceptible to infections, especially bacterial pneumonias, and that these infections are likely to be more severe than in non-alcoholics.[1–4] Various host factors present in the alcoholic patient have been implicated, including poor nutrition, environmental exposure to cold, central nervous system depression and consequent aspiration, the presence of intercurrent alcohol-related illness, and abnormal immune or inflammatory response.[1,2] In vitro and in vivo studies in nonhuman species have demonstrated the depressive effect of alcohol on leukocyte response and of acute alcohol excess on the host inflammatory response to infection.[5,6] The mechanisms at play in the chronic alcoholic patient responsible for heightened susceptibility to infection have not been defined, however. Prior to the antibiotic era, chronic alcoholism was demonstrated to be associated with a high incidence of and increased mortality from bacterial pneumonias,[7,8] but no epidemiologic studies had been undertaken since the introduction of antibiotic agents to confirm these findings or to correlate the occurrence of pneumonia with various host factors in the alcoholic population. This report describes a retrospective study of all alcoholic and pneumonia patients admitted to Freedmen's Hospital in 1 year. The clinical, laboratory, and epidemiologic features of alcoholic patients with and without pneumonia and of non-alcoholic patients with pneumonia were compared.

Case Selection

The study population was drawn from adults aged 20 years or older admitted to Freedmen's Hospital during the period July 1, 1966 through June 30, 1967. Freedmen's Hospital is a 439-bed general teaching hospital, serving a predominantly middle and lower socioeconomic population. Charts of those patients with the primary or secondary discharge diagnoses of pneumonia and/or alcoholism or alcohol-induced illness were reviewed. Patients with any of the following discharge diagnoses were included in the study: Pneumonia, acute brain syndrome associated with alcohol intoxication, chronic brain syndrome associated with alcohol intoxication, alcohol addiction, alcoholism, adverse effects, cirrhosis of the liver with alcoholism, and Wernicke's encephalopathy. In addition, charts with a discharge diagnosis of acute or chronic pancreatitis, gastritis or duodenitis,- beriberi, malnutrition, or epilepsy were reviewed; patients with any history of alcoholism were included in the study.

Diagnostic Criteria

Patients were divided into 3 major groups; non-alcoholics, alcoholics with pneumonia, and alcoholics without pneumonia. Alcoholism was defined by the following criteria:

1. History of regular consumption of a half pint or more of wine or hard liquor or more than 12 bottles of beer per day for a prolonged period of time.
2. Evidence of organic disease during the admission attributed by the examining physician at Freedmen's Hospital to alcohol excess, with or without a history of heavy alcohol consumption.

Four patients who showed no evidence and gave no history of chronic alcoholism had recently consumed large quantities of alcohol and were thus classified as acute alcoholics only. The diagnosis of pneumonia was accepted if there were physical signs of consolidation or the presence of an infiltrate on a chest X-ray film and a clinical course suggesting an acute infectious process of the lungs. In the absence of these criteria an autopsy report of pneumonia alone was acceptable. Tuberculous and possible viral, mycoplasmal, or fungal pneumonias were not excluded; only 1 tuberculous pneumonia could be documented with the information available in the charts. Of 183 charts with the discharge diagnosis of pneumonia 121 (66.1%) met these criteria.

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The charts that met the above criteria were reviewed, and the history of alcohol consumption, description of the patient's clinical course, laboratory test results, and epidemiologic data were excerpted.

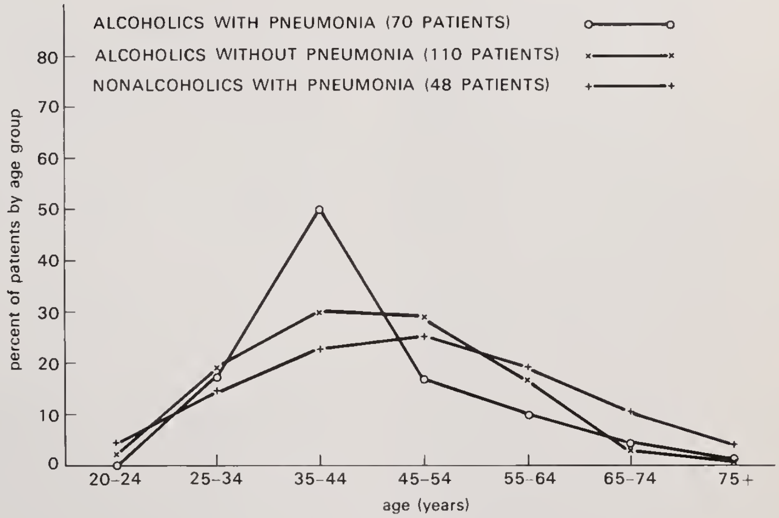

FIG. 1. Age distribution of pneumonia and alcoholic patients.

Epidemiologic Data

For the study period, 121 admissions (118 patients) met the criteria for the diagnosis of pneumonia and 197 admissions (180 patients) for the diagnosis of alcoholism. Seventy-one of 197 alcoholic patient-admissions (36.0%) were associated with pneumonia. Seventy-one of 121 pneumonia admissions (58.7%) were of alcoholic patients. Because of insufficient diagnostic workup for pneumonia in patients on the surgical services, only 1 surgical patient met the criteria for the diagnosis of pneumonia. All other pneumonia patients had been admitted to the medical or neuropsychiatric wards. During this period there were 8,628 admissions to Freedmen's Hospital of patients over 19 years old; therefore, 1.4% of all admissions were for pneumonia, and 6.4% were of alcoholic patients; 6.4% of medical and neuropsychiatric admissions were for pneumonia.

The age distribution of the 3 patient groups is shown in figure 1. Of the 70 alcoholics with pneumonia, the sex ratio of women to men was 1:5.3, and of the 110 alcoholics without pneumonia 1:3.3. For non-alcoholics with pneumonia it approached unity 1:1.2. The mean ages of alcoholics with pneumonia, 43.2 years, and of alcoholics without pneumonia, 44.2, were not statistically different but were lower than the age of non-alcoholics with pneumonia, 48.4. The age ranges were 25-76 among alcoholics with pneumonia, 23-78 for alcoholics without pneumonia, and 23-97 for non-alcoholic pneumonia patients. There was a striking peak of alcoholic pneumonia patients in the 35-44 age group (50% of all alcoholics with pneumonia), whereas non-alcoholic pneumonia patients and alcoholics without pneumonia had a more even distribution, with most in the 35-54 age range. There were few alcoholics over age 64 (8 out of 180 or 4.4%), but 14.6% of non-alcoholic pneumonia patients were in this age group.

Admissions by month are shown in Table 1. Peak admissions of alcoholics without pneumonia and non-alcoholics with pneumonia occurred in the summer, and there was a slight dropoff of admissions of non-alcoholic pneumonia cases in the autumn and early winter. The number of cases is too small for these trends to be significant.

TABLE 1: Admissions of Study Patients by Month

Month	Alcoholics With Pneumonia	Alcoholics Without Pneumonia	Non-Alcoholics With Pneumonia
January	4	10	4
February	6	10	4
March	10	10	6
April	4	8	6
May	6	11	3
June	4	7	4
July	7	11	3
August	8	16	8
September	5	14	6
October	5	12	2
November	9	7	1
December	3	10	3
Total	71	126	50

The mean and median length of hospital stay for the three patient groups is shown in Table 2. Most of the

TABLE 2: Duration of Hospitalization and Fever of Study Patients

	Alcoholics With Pneumonia	Alcoholics Without Pneumonia	Non-Alcoholics With Pneumonia
Median hospital stay	16 days	14.5 days	16.5 days
Mean hospital stay	18.9 days	18.9 days	20.2 days
Mean hospital stay excluding deaths	21.1 days	18.7 days	21.8 days
Mean days initial fever	3.8 days	–	2.4 days
Mean days initial fever excluding deaths	4.1 days	–	2.3 days

admissions were for less than 3 weeks. The median hospital stay was about the same for all pneumonia patients; for alcoholics without pneumonia the median hospital stay was shorter. The mean duration of persistent temperature elevation subsequent to the diagnosis of pneumonia is also shown in table 2. Fever persisted longer in alcoholics than in non-alcoholics. Counting only those patients who recovered, the difference was greater, 4.12 days for alcoholics versus 2.27 days for non-alcoholics.

Overall death rates for each patient group and age-specific death rates are shown in table 3. The overall death rate in the alcoholic patients was 9.1%, but for alcoholics with pneumonia the mortality rate was 19.7% compared with only 3.1% for alcoholics without pneumonia. However, the death rate for pneumonia in non-alcoholics was also high, 14.0%. The average ages at death are also shown in table 3. Note the high pneumonia mortality rate for the 35-44 age group of alcoholics and the progressive increase in mortality with age in the non-alcoholic pneumonia group. The mortality rates for males and females were equal for alcoholics with and without pneumonia (20.3%, 18.2%, 3.6%, and 3.8%, respectively), but the rate was much higher for female than for male non-alcoholics (27.3% versus 3.8%).

TABLE 3: Death Rates of Study Patients by Age

	Alcoholics With Pneumonia			Alcoholics Without Pneumonia			Non-Alcoholics With Pneumonia		
Age	Cases	Deaths	Rate	Cases	Deaths	Rate	Cases	Deaths	Rate
20–24	0	0	0	2	0	0	2	0	0
25–34	12	1	8.3	22	0	0	7	0	0
35–44	36	7	19.4	33	2	6.1	13	1	7.7
45–54	12	2	16.7	42	1	2.4	12	2	16.7
55–64	7	1	14.3	23	0	0	0	2	22.2
65–74	3	2	66.7	3	1	33.3	5	2	40.0
75+	1	1	100.0	1	0	0	2	0	0
Total	71	14	19.7	126	4	3.1	50	7	14.0
Mean age of fatal cases		48.7 yrs.			48.0 yrs.		58.3 yrs.		

Death Rates by Groups

All alcoholics	18/197	9.1%
All pneumonias	21/121	17.4%
Alcs. with pneumonia	14/71	19.7%
Nonalcs. with pneumonia	7/50	14.0%
Alcs. without pneumonia	4/126	3.1%

Alcoholic History

A history of alcohol consumption habits was totally lacking in 11% of the alcoholic pneumonia patients and 6% of the non-pneumonia alcoholics. Their classification as alcoholics was based on the presence of organic disease which was attributed by the physician in charge to alcohol.

Many records failed to specify the amount of alcohol consumed; they described the patient only as a "heavy" or "chronic" alcoholic. Therefore, it is not possible to make valid comparisons of the amount of habitual alcohol consumption in the 2 alcoholic groups. However, a slightly higher percentage of the alcoholics with pneumonia, for whom alcohol consumption was stated, drank 1 or more pints of hard liquor a day than alcoholics without pneumonia 70% versus 61%. Duration of alcoholism, pattern of alcohol consumption (spree versus steady), and type of alcohol consumed were also not specified in enough charts to make comparison valid (table 4).

Clinical History

Depressed and altered state of consciousness, aspiration, and poor nutrition have been suggested as factors that predispose alcoholics to pneumonia.[1] Table 5 compares the 3 patient groups by state of consciousness and mentation at the time of admission or just prior to the onset of pneumonia. In cases in which the mental state was not specified in the history, patients were able to give a

TABLE 4: Alcohol Consumption Habits of Study Patients

Daily Intake	Alcoholics With Pneumonia	Alcoholics Without Pneumonia
> ½ Pint, < 1 Pint	12 (30%)	22 (39%)
1–2 Pints	26 (65%)	33 (58%)
> 2 Pints	2 (5%)	2 (3%)
Unspecified	30	53
	70	110
Type of Alcohol		
Hard Liquor	19 (73%)	24 (80%)
Wine	4 (15%)	6 (37%)
Beer	3 (12%)	0 (0%)
Unspecified	44	80
	70	110
Duration of Alcoholism		
1 Year	0 (0%)	1 (3%)
1–10 Years	5 (36%)	12 (37%)
11–20 Years	4 (28%)	10 (31%)
> 20 Years	0 (0%)	4 (12%)
"Many" years	5 (36%)	5 (16%)
Unspecified	56	78
	70	110

history, and we may assume they were at least alert if not sober. The unconscious group contained patients who were post-ictal, in alcoholic stupor, or comatose for other reasons. In both groups of pneumonia patients, the majority were alert and sober at the onset of pneumonia, but only 39.0% of alcoholics without pneumonia were alert and sober at the time of admission. In particular, the group of alcoholics with pneumonia had a smaller proportion of patients in impending or actual delirium tremens (23.0%) than the alcoholics without pneumonia 44.0%. Only 24.0% of alcoholics with pneumonia had objective evidence of acute alcoholism (impending or actual DT's, drunkenness, "rum fits", etc.), whereas 54.0% of alcoholics without pneumonia showed such evidence.

Descriptions of the nutritional status of most patients consisted of statements such as "well", "fairly well", or "poorly" nourished. Only 2 instances of physical findings specifically related to nutritional deficiency were noted, both among alcoholics with pneumonia, 1 of whom had a smooth, atrophic tongue and the other pellagra dermatitis. "Poorly nourished" in general implied underweight. Similar proportions of the alcoholics with and without pneumonia were described as poorly nourished, but a higher percentage of non-alcoholics with pneumonia were obese (table 5).

Smoking habits of the 3 patient groups are also compared in table 5. Since there is a larger percentage of moderate smokers (1-2 packs a day) in the alcoholics without pneumonia than in the group with pneumonia, heavy smoking did not correlate directly with the development of pneumonia. In the non-alcoholic group 90% smoked no cigarettes or less than 1 pack per day.

Table 6 lists the diagnoses by category for all 3 patient groups. Diagnoses other than pneumonia and alcoholism were discharge diagnoses or mentioned in the discharge summary. No attempt was made to substantiate physicians' diagnoses. In the case of Laennec's cirrhosis the charts were often lacking in laboratory and clinical information necessary to confirm the diagnosis.

Thirty % of alcoholics with pneumonia showed evidence of recent excessive alcoholic intake. However, 70% of alcoholic patients without pneumonia were acutely alcoholic. The incidence of DT's and seizures was also much higher in the non-pneumonia group. The incidence of cirrhosis or acute alcoholic liver disease was 18% in both groups of alcoholics.

The vast majority of pneumonia patients recovered without complications secondary to pulmonary infection. Five patients in both groups developed 1 of the following complications: Gram-negative superinfection, atelectasis, pleural effusion, empyema, cavitation, or abscess.

There were 71 admissions of alcoholics with pneumonia; in 67 cases pneumonia was present on admission. Aspiration pneumonia developed in 3 of the other 4 patients with hepatic coma, postictal state, severe third degree burns, and diabetes with a subcutaneous abscess. Of the 50 non-alcoholic pneumonia admissions, pneumonia was diagnosed on admission in 47 cases. The 3 who acquired pneumonia after admission were a postoperative gynecology patient, a man with an intracerebral hemorrhage, and a patient with terminal pancreatic carcinoma; they were also thought to represent aspiration pneumonia.

An analysis of the deaths by associated illnesses reveals the following. Of the 7 non-alcoholic pneumonia patients who died, 5 were moribund

TABLE 5: State of Consciousness,* Nutritional Status and Smoking Habits of Study Patients

State of Consciousness	Alcoholics With Pneumonia	Alcoholics Without Pneumonia	Non-Alcoholics With Pneumonia
Alert, sober	48 (70%)	41 (39%)	42 (93%)
Unconscious	4 (6%)	6 (6%)	4 (8%)
Impending DT's	15 (22%)	29 (28%)	0
DT's	1 (1%)	17 (16%)	0
Chronic brain syn.	0	1 (1%)	0
Drunk	1 (1%)	10 (10%)	0
Unspecified	2	22	4
	71	126	50
Nutritional State			
Obese	2 (3%)	2 (2%)	8 (18%)
Well nourished	42 (64%)	74 (73%)	27 (60%)
Fairly well nour.	5 (8%)	2 (2%)	1 (2%)
Poorly nourished	17 (26%)	23 (23%)	9 (20%)
Unspecified	4	9	3
	70	110	48
Smoking Habits			
None	9 (14%)	16 (19%)	13 (30%)
1 pack	40 (63%)	36 (42%)	26 (60%)
1-2 packs	13 (21%)	32 (37%)	3 (7%)
"Heavy"	1 (2%)	2 (2%)	1 (2%)
Unspecified	7	24	5
	70	110	48

* At the time of admission or of diagnosis of pneumonia.

on admission and had a terminal illness—subarachnoid hemorrhage, carcinoma of the esophagus, adenocarcinoma of the pancreas, intracerebral hemorrhage, or myocardial infarction with pulmonary edema. One patient had a cerebrovascular accident with hemiparesis, and another had diabetes mellitus with a subcutaneous suprapubic abscess. Four alcoholics without pneumonia died, 3 with severe cirrhosis and 1 with acute pancreatitis and delirium tremens. Five of the 14 alcoholics with pneumonia who died had evidence of Laennec's cirrhosis or acute fatty liver, and 2 had acute pancreatitis. Three had delirium tremens. The 2 lowest white blood cell counts were in alcoholics who died (1,200 and 1,900); this has been noted to be a poor prognostic sign.[2] The fatal cases are described in table 7.

Laboratory Data

X-ray films or X-ray reports were available for approximately 80% of admissions of pneumonia patients. In alcoholics and non-alcoholics the lower lobes were involved most frequently, 84% and 80%, respectively. Left-sided and bilateral involvement was somewhat more common in the alcoholics with pneumonia than in the non-alcoholics, but in both groups right-sided involvement was most common, 44% and 63%, respectively.

Sputum culture results were available for 38 of 70 alcoholics and 20 of 50 non-alcoholics (table 8). Pneumococci were isolated from 14 patients, Klebsiella from 7, and *Staphylococcus aureus,* coagulase-positive from 1. The most common organism cultured was alpha streptococcus, the significance of which as a pathogenic organism is questionable because of the lack of positive blood cultures or adequate sputum smears. Tubercle bacilli were recovered from 1 alcoholic with pneumonia and 2 alcoholics with no pulmonary infiltrate but with cavitation. In general, descriptions of Gram stains were inadequate in that the organisms were rarely described in detail and the numbers and types of cellular elements were not well described.

Blood cultures were taken from the majority of pneumonia patients with high fever. Blood cultures were positive in 7 cases: 5 D. *pneumoniae,* 1 *S. aureus,* coagulase positive, and 1 Klebsiella. The source of sepsis was thought to be the lung in all cases.

The mean and median values of the total white blood cell count and % of polymorphonuclear leukocytes and results of various liver function tests at admission or the time of diagnosis of pneumonia are presented in table 9. The liver function tests were performed for so few patients that it is impossible to draw conclusions regarding the severity of liver abnormality in the various groups. As expected, the mean and median WBC for the 2 groups with pneumonia was significantly higher than for the non-pneumonia group. However, alcoholics, especially those with pneumonia, had the lowest white counts.

TABLE 6: Other Diagnoses of Pneumonia and Alcoholism Patients

Alcohol-related	Alcoholics With Pneumonia	Alcoholics Without Pneumonia	Non-Alcoholics With Pneumonia
Acute and chronic alcoholism	20 (30%)	74 (70%)	0
Acute Alcoholism only	1	3	0
Chronic alcoholism only	49	33	0
Impending DT's	5	12	0
DT's	11	42	0
Alcoholic stupor	2	1	0
Neurologic			
Seizure disorder	2 (3%)	23 (21%)	3
Korsakoff syndrome	0	2	0
Chronic brain syndrome	0	8	0
Neuropathy	0	6	0
Gastrointestinal			
Cirrhosis or alcoholic liver disease	13 (18%)	20 (18%)	0
Pancreatitis (Acute or Chronic)	5 (7%)	11 (10%)	0
Gastritis	2	6	0
Peptic ulcer	2	3	0
Hepatic coma	1	0	0
Cardio-respiratory			
TBC (active)	1	2	0
P.M.D.	0	1	0
Asthma	1	1	3
Bronchitis	2	2	4
R.H.D.	1	0	0
H.A.S.H.D.	3	0	4
Pleural effusion or empyema	4	0	1
Emphysema	0	0	1
Lung abscess	1	0	0
Malignancy	0	0	4
Diabetes	7	0	2
Trauma or burns	1	14	0
Pellagra	1	0	0
Anemia	4	4	3

Discussion

Beginning with Koch's observation in 1884 that alcoholics were particularly susceptible to the ravages of epidemic cholera, numerous investigators have noted the heightened susceptibility of alcoholics to various types of infections.[6] Osler[8] wrote about pneumonia that "the most potent predisposing factor is perhaps the lowered resistance due to alcohol". Capps and Colemann[7] retrospectively studied 3,422 pneumonia cases reported in Chicago in 1911-1922 and found that mortality increased in proportion to the amount of alcohol consumption for any particular age range. Overall, excessive drinkers had a mortality rate of 49.9%, modest drinkers 34.4%, and abstainers 22.5%. No epidemiologic data are available, however, on pneumonia morbidity or mortality in acute versus chronic alcoholics.

Prior to the antibiotic era there were many articles on the depressive effect of alcohol on host-immune and inflammatory response.[5,6] Lushbaugh[5] recommended giving larger doses of antipneumonococcal serum to alcoholic patients because he had found out that larger doses in intoxicated rabbits compensated for the impaired leukocyte response. This protective effect has been demonstrated in vitro and in vivo with various animal species. These experiments are summarized in a comprehensive review by K. L. Pickrell published in 1943.[6] He concluded that alcohol intoxication to the point of stupor destroys resistance to pneumococcal infection in the rabbit by inhibiting leukocyte migration to the site of infection. The mechanisms which might predispose to pneumonia in the chronic alcoholic who is not acutely intoxicated are not, however, defined.

With the introduction of antibiotics and the consequent decline in mortality from pneumonia, interest in the association of pneumonia with alcoholism and in the mechanisms involved in altering host resistance in the alcoholic waned. Yet, alcoholics continue to make up a large proportion of pneumonia patients, and pneumonitis continues to be a leading cause of hospitalization among alcoholics. Nolan's study[10] of 900 consecutive medical admissions to Grace New Haven Community Hospital showed that 13.8% of these admissions were of alcoholics and that 8% of all patients admitted had pneumonia, but that 16.9% of alcoholics and only 6.5% of non-alcoholic patients had pneumonia.

TABLE 7: Deaths: Pneumonia And Alcoholism Study Patients

Age	Sex	Associated Diagnoses	Other
		Alcoholics with Pneumonia	
66	M	Lung abscess, chronic alcoholic	Heavy Smoker
44	M	DT's, aspiration pneumonia, chronic alcoholic	Poorly nourished
37	F	Cirrhosis; anemia	Poorly nourished
68	M	Acute pancreatitis; diabetes mellitus	
42	M	Severe 3° burns, acute alcoholism	
49	M	DT's; pellagra dermatitis; cirrhosis	Hospital-acquired pneumonia
76	M	Cirrhosis, post-ictal	
29	M	Acute and chronic alcoholism, fatty liver	
36	M	DT's, pancreatitis, hypertension, aspiration pneumonia	
59	M	Aspiration pneumonia; alcoholic stupor	Poorly nourished
44	F	Fatty liver, leukopenia	Poorly nourished
54	M	Acute and chronic alcoholism	
37	M	Leukopenia	Poorly nourished
41	M	Anemia; emphysema; congestive heart failure	Poorly nourished
		Alcoholics Without Pneumonia	
35	F	Cirrhosis; ascites; TBC; anemia	Poorly nourished
69	M	Cirrhosis, acute and chronic alcoholism DT's; primary myocardial disease; hepatic coma.	
42	M	Acute pancreatitis, DT's, acute and chronic alcoholism	
46	M	Acute and chronic alcoholism; cirrhosis; seizures	
		Non-Alcoholics With Pneumonia	
68	F	Subarachnoid hemorrhage; hypertension	
52	F	Carcinoma of esophagus	
71	F	Acute myocardial infarction; pulmonary edema	
53	F	Old CVA; aspiration pneumonia	
58	F	Intracerebral hemorrhage	Hospital-acquired pneumonia
62	M	Adenocarcinoma of pancreas with metastases;	Hospital-acquired pneumonia
44	F	Diabetes; obesity; suprapubic abscess	

Pneumonia was the most common primary diagnosis for alcoholics admitted. The mortality rate for alcoholics was 19.4% versus 13.2% for non-alcoholics.

Our study was undertaken to obtain epidemiologic data on the occurrence of pneumonia today in an urban, general hospital population and to determine the mortality and clinical setting of pneumonia in alcoholic and non-alcoholic patients. The limitations imposed by retrospective study of a small selected population make detailed analysis of multiple host variables impossible and generalization of the findings to the entire alcoholic population hazardous. Nolan[10] noted the difficulties of obtaining reliable alcoholic histories even in a prospective study. In our study charts often lacked detailed descriptions of the pattern of excessive drinking habits. Nevertheless, patients could be classified as alcoholic or non-alcoholic, and acute alcoholism could be distinguished from chronic. Undoubtedly, some less severely alcoholic patients who showed no signs of organic alcohol-induced disease were overlooked. However, the observations do show the clinical setting in which pneumonia occurs among alcoholics.

The findings confirm that alcoholics continue to constitute a sizeable portion of admissions in this hospital and that pneumonia is the leading cause of hospitalization for alcoholics. More than half of all adult pneumonia patients in the study were alcoholics. The male preponderance of alcoholic patients probably reflects the true preponderance of men in the alcoholic community from which the hospital draws its patients, but no study is available of the age and sex distribution of alcoholics in the District of Columbia for comparison. Studies of alcoholic populations in other cities[13,14] indicate a female-to-male ratio of 1:2 to 1:8 for nonwhites and 1:3 to 1:8 for whites. In a study of 427 pneumonia patients, both alcoholics and non-alcoholics, at the District of Columbia General Hospital[11] the sex ratio was similar, 1:2.1 versus 1:2.6 in our study. The difference in male-to-female sex ratio among alcoholics with and without pneumonia, 1:5.3 versus 1:3.2, was not significant ($P < .10$), but the difference in sex ratios between alcoholics and non-alcoholics with pneumonia was significant ($P < .00025$).

The age distribution of total pneumonia patients also agrees closely with that found in the D.C. General Hospital study. However, alcoholics with pneumonia were generally younger than non-alcoholics with pneumonia, reflecting the frequent occurrence of pneumonia as a complication of other chronic or terminal illness in non-alcoholics. The age distribution for alcoholic patients without pneumonia does not show this marked predilection for the younger age groups (figure 1).

There was no significant seasonal variation in the occurrence of pneumonia in this study, although admissions of alcoholics without pneumonia peaked in August. There is no evidence from our data that exposure to cold during the winter months resulted in any increase in pneumonia admissions.

The severity of pneumonia was judged by the mortality rate, duration of fever after the diagnosis of pneumonia, and duration of hospital stay, although concurrent illnesses and other factors would, of course, also influence these parameters. The overall mortality rate for alcoholics with pneumonia, 19.7%, was greater than for non-alcoholics with pneumonia, 14.0%, and much higher than for alcoholics without pneumonia, 3.1%. These differences are all significant. Thus, in this hospital and in this antibiotic age, the pneumonia death rate is still high, and alcoholics with pneumonia have a significantly greater risk of death. The presence of pneumonia greatly increased the risk of death for alcoholic patients in our study. This difference between mortality rates for alcoholics and for non-alcoholics was most striking in the younger-age groups.

TABLE 8: Sputum and Blood Cultures of Study Patients

Organism	Alcoholics	Non-Alcoholics With Pneumonia
Sputum		
D. Pneumoniae ± other organisms	9	5
K. Aerobacter ± other organisms	4	3
S. Aureus Coagulose + ± other organisms	1	0
Streptococci ± other organisms	21	10
Multiple other organisms	3	2
No culture reported	33	30
	71	50
Blood		
D. Pneumoniae	2	3
K. Aerobacter	1	0
S. Aureus Coagulose +	0	1

The mean duration of persistent temperature elevation subsequent to the diagnosis of pneumonia was 2 days longer for alcoholics than for non-alcoholics. This may reflect the presence of other fever-producing illness, such as DT's, seizures, or pancreatitis, rather than a slower response to treatment of the pneumonia or greater severity of infection.

The presence of pneumonia in hospitalized alcoholics did not correlate with the presence of alcoholic liver disease or of altered state of consciousness due to the effects of alcohol. However, patients with other alcohol-induced diseases died of pneumonia, but non-alcoholics with pneumonia usually died of another terminal illness which was complicated by pneumonia.

Not enough information was available to relate the amount, type, duration, or pattern of drinking to the occurrence of pneumonia. Where data were available there appeared to be no significant correlation between type of alcohol habitually consumed or amount and development of pneumonia. Similarly, gross nutritional status did not correlate with the occurrence of pneumonia. Contrary to the widely held impression that depressed or altered states of consciousness, such as occur in drunkenness, DT's and the post-ictal state, predispose to pneumonia, alcoholics without pneumonia had a significantly higher incidence of these and other signs and symptoms of acute alcoholism than those with pneumonia.

For only 58 of 121 pneumonia patients were bacteriologic studies adequate. Of the adequately studied patients only 47% yielded an etiologic bacterial agent, compared with 53% of pneumonia patients from whom a bacterial etiology could be documented in the study of Mufson et al.[11] In that study an etiologic agent was found in 72% of all pneumonia patients—53% bacterial, 15% viral, and 4% mycoplasmal on the basis of serologic studies. We did not do viral and mycoplasmal studies, but the clinical course of our study patients was not inconsistent with a viral or primary atypical pneumonia in many cases.

The need for a prospective study involving larger numbers of patients is apparent if one is to obtain detailed enough epidemiologic information to correlate the occurrence of pneumonia with immunologic, biochemical, or physiologic defects in the alcoholic patient. This study points up, however, that it is not alcohol intoxication and a high blood or tissue level of alcohol that predisposes to pulmonary infection, but that some mechanism at work in the chronic but not acutely alcoholic patient also makes him more

TABLE 9: Laboratory Data for Study Patients

	Alcoholics With Pneumonia	Alcoholics Without Pneumonia	Non-Alcoholics With Pneumonia
	WBC		
Mean	13,580	8,550	12,260
Median	11,700	7,850	12,075
Range	1,200–46,500	2,500–37,500	3,500–31,000
No. patients	69	108	44
	SGOT		
Mean	66.2	75.0	42.8
Median	67.0	66.0	32.0
No. patients	26	44	17
	Alk. phosphatase		
Mean	3.01	2.85	3.47
Median	3.58	2.30	3.00
No. patients	29	43	13
	Bilirubin		
Mean	2.08	1.57	2.11
Median	1.32	0.97	0.67
No. patients	26	36	12
	A/G Ratio		
No. patients with A/G 1	28 (70%)	46 (78%)	21 (81%)
No. patients with A/G 1	12 (30%)	13 (22%)	5 (19%)

susceptible to pneumonia. This mechanism might be one of diminished immune response systemically or locally in the lung parenchyma, or impaired inflammatory response. Our findings among hospitalized alcoholics of a lack of correlation of the development of pneumonia with state of consciousness, nutrition, presence of liver disease, old age, or sex should not be interpreted to mean that these factors have no role in predisposing to pulmonary infections. Hospitalized alcoholics are a selected group representing those most severely or acutely affected by organic and psychic damage from alcohol. If one could obtain similar data on the entire alcoholic population, the incidence of pneumonia would most likely be greatest for those with depressed levels of consciousness and evidence of organic alcohol-associated disease, such as cirrhosis. A positive correlation in our study population would have been evidence of a causal relationship; however, the opposite conclusion cannot be drawn on the basis of our findings, especially in view of the small numbers of patients studied. Some other factor of chronic alcoholism may predispose alcoholics to pulmonary infections, and altered consciousness and hepatic disease may be only independent factors prevalent in the alcoholic population that becomes hospitalized. Further work needs to be done to clarify this.

Summary

The study consisted of a retrospective survey of all adult alcoholics and non-alcoholics with pneumonia admitted to Freedmen's Hospital over a 1-year period. More than half of all pneumonia patients admitted were alcoholics, and pneumonia was the leading cause of hospitalization for alcoholics. Alcoholics with pneumonia were primarily in the younger age groups, but non-alcoholics with pneumonia were of all ages. The death rate for all pneumonia patients was low compared with the pre-antibiotic era but was slightly higher for alcoholics. Pneumonia increased the risk of death greatly for hospitalized alcoholics. Acute alcoholism, depressed mental state, poor nutrition, and the presence of alcoholic liver disease did not correlate directly with the presence of pneumonia in hospitalized alcoholics in our series. Not enough data were available to correlate the amount or type of alcohol consumed with pneumonia morbidity or mortality. A prospective study of a larger patient population may point out a correlation between certain physiologic, immune, or biochemical defects and susceptibility to pneumonia in the alcoholic.

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Exogenous Lipoid Pneumonia*
Report of Three Cases with Selective Review of the Literature

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Since the animal experiments of Laughlen[1] in 1925 and Pinkerton[2] in 1927, showing serious pathologic changes in the lungs by chronic introduction of mineral oil into the nose or throat,[3] it has been repeatedly demonstrated that chronic ingestion of mineral oil, either as a laxative or as nasal medication over a period of time, can produce pneumonitis in children and in debilitated, recumbent, aged persons. It can also occur in healthy ambulatory adults. Progression of disease is so insidious that diagnosis is extremely difficult, unless the possibility is kept in mind.

We are reporting 3 cases of exogenous lipoid (oil) pneumonitis.

Case 1. C. N., FH #86-282-A, a 64-year-old retired college professor was first admitted to the pulmonary disease service in October, 1962 with symptoms of wheezing 2 days prior to admission. One week later he developed right-sided pleuritic pain followed by nonproductive cough, chills, fever, and night sweats. He had taken 3 tablespoonsful of mineral oil nightly for about a year for chronic constipation. He was hypertensive and had suffered a cerebrovascular accident for which he was hospitalized in 1952.

On admission the blood pressure was 142/74, the temperature 102° F, the pulse rate 104 per minute and regular, and the respiratory rate 32 per minute. Emotional instability was manifested by crying, and there was a residual neurological deficit involving IX and X cranial nerves with right-sided hemiparesis. The chest showed diminished respiratory excursions bilaterally, with wheezes and crackling rales, both anteriorly and posteriorly in the mid-lung fields. There was hepatomegaly with smooth nontender border and a moderately enlarged prostate gland.

The white blood cell count was 15,000 with shift to the left. The hemoglobin was 10.6 Gm and the hematocrit 37%. Skin tests were negative for tuberculosis, histoplasmosis, and coccidioidomycosis. A Gram stain showed "mixed flora". A sputum culture for *M. tuberculosis* was negative, but a Sudan III stain of the sputum was positive for oil.

The patient was placed on tetracyclines. Diagnostic bronchoscopy revealed reddened granular mucosa with mucoid secretions from the lower lobes. Bronchial washings were negative for malignant cells but were positive for acid-fast bacilli by smear and *M. tuberculosis* by culture. While in the hospital the patient was inadvertently placed on mineral oil nightly for constipation. The nurses' note read, "Mineral oil omitted due to oozing of oil from the rectum". An esophagram was negative. Pulmonary function tests showed a restrictive spirographic pattern. The patient was placed on antituberculous chemotherapy, along with other symptomatic treatment, but the cough continued with bouts of hemoptysis. He lost 8 to 10 pounds within a few weeks and developed insidious clubbing. Right pleural biopsy was done followed by scalene node biopsy 4½ months later.

Sections of the pleural biopsy showed fragments of fibrocollagenous tissue and some skeletal muscle. The former apparently represented pleura, and attached to it was a rim of collapsed lung. The pleura was irregularly thickened and partially covered by organizing fibrin clots, and in areas infiltrated with histiocytic cells and lymphocytes. The alveolar septa were focally thickened and fibrosed. Although the air spaces were generally collapsed, occasional alveoli contained lipoidal inclusions, together with some macrophages (fig. 1a).

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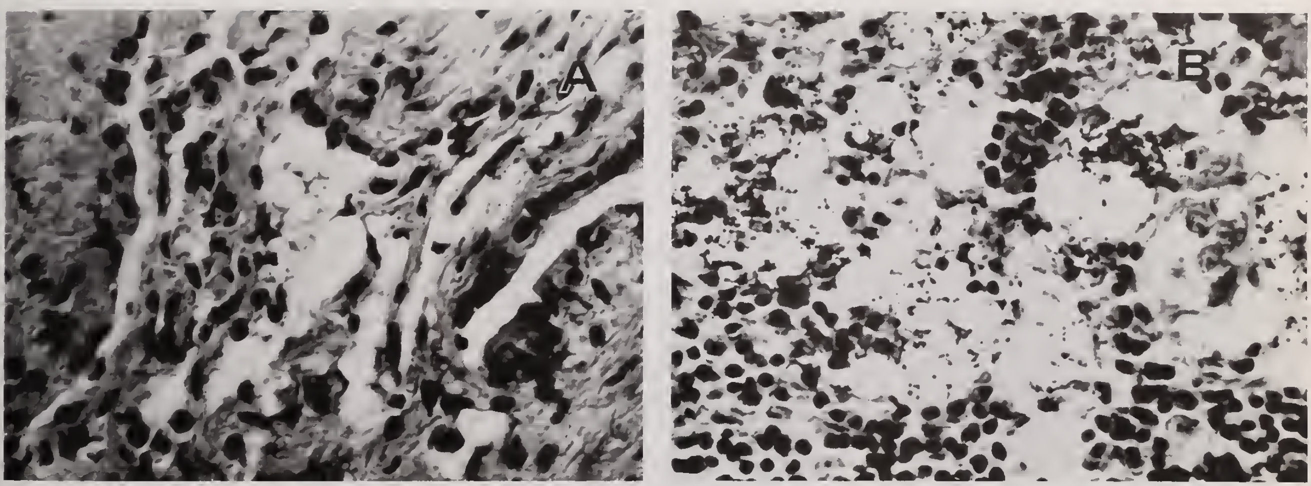

FIG. 1A. Case 1. Lung showing lipid inclusions and some mononuclear cells in the collapsed alveoli (bronchiole on right). Pulmonary fibrosis is evident. 1B. Case 1. Scalene lymph node with lipoid granulomatosis. Asteroid body in a giant cell in the right mid-field. Granules admixed with lipid material represent anthracoctic pigment.

Sections of 5 scalene lymph nodes, taken 4½ months later, showed essentially similar findings, although their degree varied. The nodes were scattered with lipophages and anthracotic pigment-laden macrophages which were often aggregated to form ill-defined nodules or in a geographic pattern. Mingled in these macrophages were lipid vacuoles and multinucleated giant cells. Occasionally an asteroid body was found within the giant cells (fig. 1b). Some of the less vacuolated histiocytes were also laden with yellowish brown, somewhat refractile granules, resembling those of ceroid.

Comment

There are several points worth noting in this case. There was failure to illicit a history of oil ingestion, but the clue to the diagnosis was suggested by reading the nurses' notes. The importance of cranial-nerve involvement and defective swallowing mechanism should be noted. The nodular pulmonary infiltrate, which suggested lung cancer, was shown to be a paraffinoma and, finally, the association of tuberculous infection with this patient's illness.

Case 2. L.M.H., FH #308-424, a 26-year-old, licensed practical nurse, was admitted to Freedmen's hospital on November 11, 1970 because of progressive cough and dyspnea, accompanied by low back pain. In January 1967 she had Klebsiella pneumonia, and in October she was treated for pulmonary embolism in another hospital. Because of persistance of the pulmonary infiltration she was re-admitted in March 1968 with the diagnosis of chronic bronchitis, with suspicion of sarcoidosis. She had a history of excessive castor oil ingestion as a child, which suggested the possibility of lipoid pneumonia.

Physical examination revealed a thin young woman, with moderate clubbing of the digits. The blood pressure was 110/68, the pulse 80 per minute and regular, the respiratory rate 26 per minute, and the temperature normal. There was a right-sided healed anterior thoractomy scar and bibasilar inspiratory crackling rales.

The skin tests were negative for tuberculin, histoplasmin, and cocidioidin. The white blood cell count was 13,000. Plasma proteins and A/G ratio were normal. Serum calcium, alkaline phosphatase, and other liver function tests were normal. Examination of sputum was negative for acid-fast bacilli. Sputum cultures showed mixed organisms of streptococci and Neisseria species. Sputum stains for Sudan III were positive for fat. Chest X-ray film is shown in fig. 2. Pulmonary function tests are shown in table 1. Open lung biopsy was done at another institution, and the diagnosis of lipoid pneumonia was confirmed.

Sections of the lung biopsy from the other hospital showed that the pleura was irregularly thickened and focally lined by swollen mesothelial cells. Attached to the pleura were occasional small tags of fibrous tissue. The alveoli were collapsed in some areas, and in others they were emphysematous. Often, the alveolar spaces contained clumps of pneumocytes, some of which were pigmented (fig. 3), and occasional ones were vacuolated. Some were also filled with pink to brown granular material. In places the septa were thickened, fibrosed, and infiltrated by mononuclear cells. Some of the macrophages within the alveoli and septa laden with acid-fast pigment-granules. These were suggestive of a ceroid-like substance.

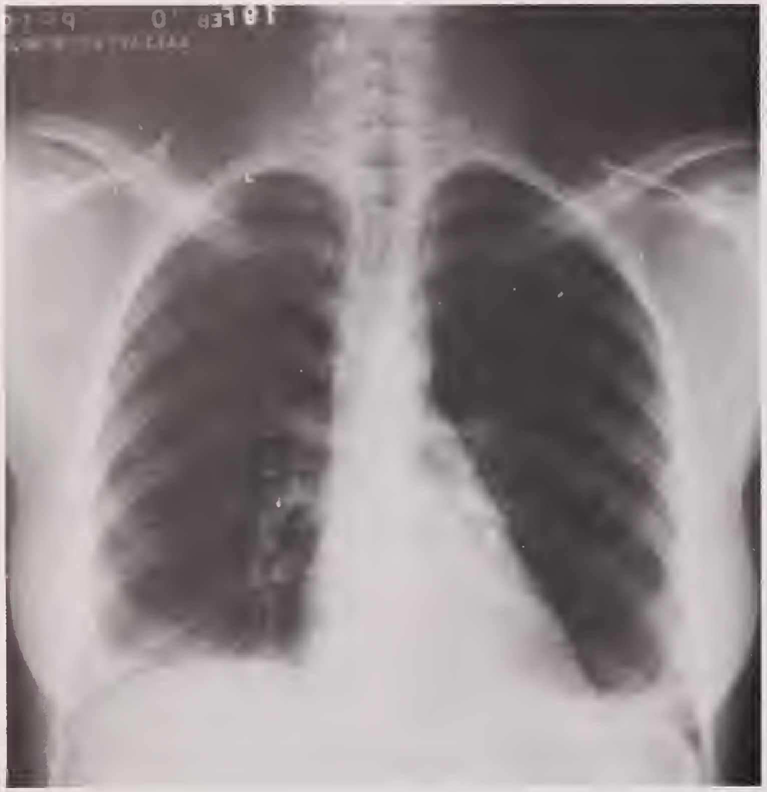

FIG. 2. Admission chest X-ray film in case 2 showing bilateral basilar linear infiltrate, more marked on right and partially obscuring the hemidiaphragms. Remainder of lung zones are clear. The pattern is reminiscent of that in desquamative interstitial pneumonia.

The patient was re-admitted to Freedmen's Hospital on January 9, 1970 because of spontaneous lower chest pain. Diagnostic bronchoscopy showed copious secretion from all orifices. Pulmonary function tests were repeated. They showed slight improvement when compared with those of February 1968. With symptomatic treatment the patient improved and was discharged in fair condition.

Case 3. L.E.W., FH #315-530, a 47-year-old black female domestic worker, came to the hospital on August 13, 1970 because of progressive dyspnea for the past few months. Cough and wheezing followed, which subsided later, but the following day she noticed progressive dyspnea on the slightest exertion. She had been unable to tolerate strong odors. She had taken petroleum jelly for colds.

Her blood pressure was 146/90, the pulse 88 per minute and regular, and the temperature 99° F. There were diminished breath sounds at both bases with some crackling rales. The remainder of the examination was essentially normal. The white blood cell count was 18,100 with a shift to the left. The hemoglobin was 12.6 gm. The red cells were hypochromic and microcytic. Sputum stains with Sudan III were positive for oil. The X-ray film is shown in figure 4. Pulmonary function tests are shown in table 2.

The patient was treated symptomatically. She had had a scalene lymph-node biopsy in another hospital, which failed to yield the diagnosis. Open lung biopsy from the anterior segment of the right upper lobe was done. Gross appearance of the resected specimen showed multiple small granules and pinkish gray moderately firm lung tissue.

Sections of the lung showed that many of the alveoli were filled with a pink granular material, clumps of foam macrophages, some multinucleated giant cells, desquamated pneumonocytes, and a few polymorphonuclear leukocytes. In places the alveolar lining cells were prominent, and the septa were thickened and scattered with mononuclear cells. Where there were aggregates of foam macrophages in the alveoli the septa often disclosed proliferation of young myxomatous fibrous tissue, together with some nodular protuberances (fig. 5a). Elsewhere there were alternating areas of atelectasis and emphysema. The pleura was focally thickened and likewise infiltrated with mononuclear cells. An oil red O stain revealed abundant lipid droplets within the macrophages and free in the alveoli (fig. 5b).

Discussion

The use of mineral oil as a laxative or of oily nose drops should arouse a high index of suspicion of lipoid pneumonia. The disease may have a varied clinical presentation with complex pathogenesis.

The response of alveoli to exposure to injury is modified by several properties of local environment of the lung as well as the nature, dose and duration of an agent which reaches the alveoli. The severity of the reaction in the lung to aspirated oil depends mainly upon its character. When saponifiable oil enters the lower respiratory tract the type of immediate reaction is due to the amount of fatty acid present or produced from the aspirated oil by tissue enzymes.[4] Mild vegetable oil, like poppy seed oil or olive oil, causes the least reaction. On the other hand, animal fats are capable of producing sudden and violent reactions, with hemorrhage and necrosis. The response to mineral oil is extremely mild, amounting to a foreign-body reaction to an inert substance within and around the alveoli in which the oil settles. In this stage the diagnosis is very difficult, unless the index of suspicion of this insidious disease is increased. It has been demonstrated that mineral oil is so bland that when introduced into the nasopharynx it finds its way into the lung without causing cough.[5] In apparently healthy persons only a minute quantity of the oil gets down into the lower bronchi. In time, some of it is fortunately removed by expectoration and to a lesser degree by phagocytosis. Its accumulation in the alveoli may possibly be augmented by inhibition of the ciliary clearance mechanism.[6] Changes in pH due to carbon dioxide retention and hypoxia due to airway obstruction can retard the delicate clearing mechanism of the lung by destroying sensitive cells and changing the cell population in alveoli.[7] Even changes in cation levels may stimulate certain cells like fibroblasts and depress the function of others.[8] Water-immiscible mineral oil may move even more rapidly because of its hydrophobic surface and surface charge, but its presence in terminal bronchioles may hinder the muco-cilliary clearance system and further contribute to the collapse of alveoli by changing the surface tension.[7]

TABLE 1: Pulmonary Function Tests (Case 2)

Static Lung Volumes	*Measured*		*Predicted*	
IC*	1.34	59%	2.26	L
ERV	0.69	61%	1.13	L
VC	2.03	60%	3.39	L
RV	1.80	130%	1.39	L
FRC	2.49	99%	2.51	L
TLC	3.83	80%	4.78	L
RV/TLC		47%		29%
End tidal CO_2	22mm Hg			
Forced expired CO_2	26mm Hg			
Arterial Blood & Gas Exchange				
	Rest	*Exercise*		
DL_{CO}	20	88%	23 ml/min/mm Hg	
Pa_{O_2}	90	90	85-95mm Hg	
Sa_{O_2}	95	95	95-100%	
Pa_{CO_2}	34	33	38-40mm Hg	
pH	7.37	7.36	7.38-7.42	
Mechanics of Breathing				
	Measured		*Predicted*	
% $FEV_{1.0}$	82		72–98%	
% $FEV_{3.0}$	100		92–100%	
MVV	69.6 (82%)		84.5 L/min	

Pulmonary Function Studies

There have been few reports of lung-function studies in oil pneumonia. Pulmonary function data in our cases revealed restrictive lung disease with varying degrees of hyperinflation of lung tissue. Data for cases 2 and 3 are shown in the table. A decreased vital capacity 50% of predicted is said to be common.[9] Restriction may be associated with significant amounts of airways obstruction, which is significant in spite of lack of changes in terminal airways microscopically and is difficult to explain.[10] Hoffard[9] tried to explain it on the basis of associated cigarette smoking, but no patient had a smoking history. Reflex bronchospasm may be a possibility.

Although the diffusing capacity was normal in case 2, it was decreased in case 3. Diffusing capacity is said to be, “usually normal, or only moderately decreased, particularly in the earlier stages of the disease,”.[9] Normoxemia was present in case 2 at rest and during exercise, but hypoxemia was present in case 3 at rest which worsened during exercise and arterial blood failed to achieve maximal oxygenation with the patient breathing 100% oxygen, documenting the gas transfer defect, in addition to venous admixture to the pulmonary circulation. Mild hyperventilation was present in both cases.

Diagnosis

Until recently, the diagnosis of lipoid pneumonia had not been suspected prior to pathological study. More attention must be paid to the history of oil ingestion, which is the most important clue for making the correct diagnosis. Radiological manifestations when present are nonspecific but in the majority of instances the lesions are bi-basal, and in

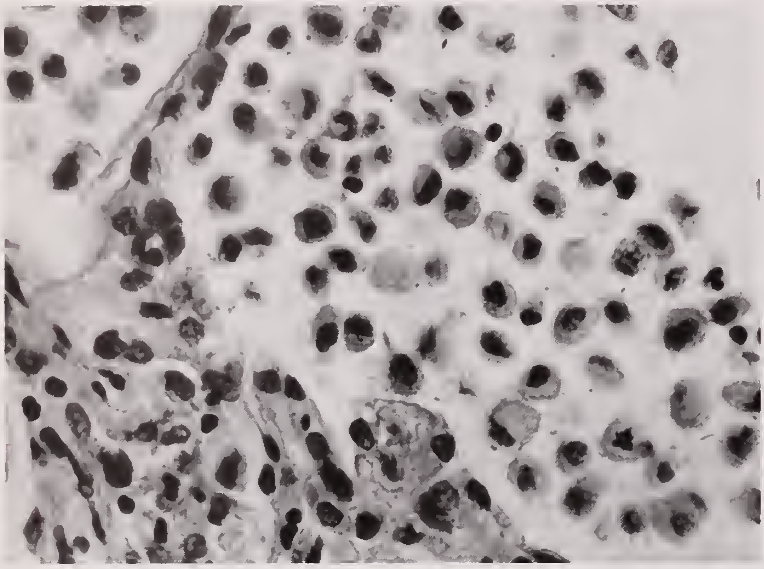

FIG. 3. Case 2. Lung biopsy showing many granular pneumonocytes in the alveoli. Some pneumonocytes are foamy; mitotic figure in the right lower field. The septum is scattered with lymphocytes and histiocytic cells.

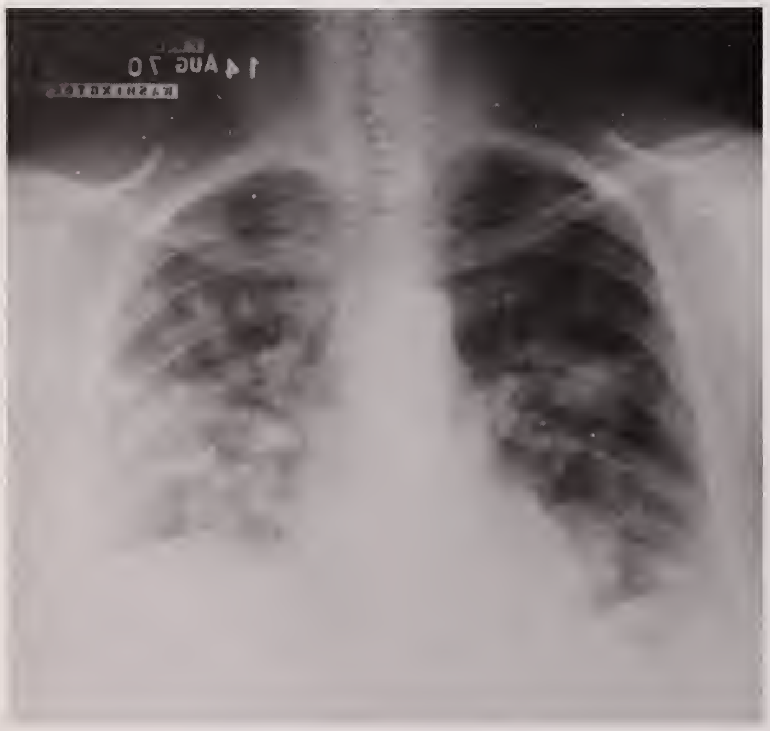

FIG. 4. Admission chest X-ray film in case 3 showing bilateral flocculo-nodular infiltrates in an "alveolar filling type pattern" more prominent in the mid and lower lung zones and on the right.

the peripheral part of the lung. The greatest help is achieved by simple sputum examination for lipophages.[11-13] Sputum examination gives more than 85% of positive results. All 3 of our patients had positive sputa for fat cells. Bronchoscopy is usually negative unless there is secondary infection. Further confirmation by lung biopsy may be helpful.

More complete and accurate identification of oil is best made by chemical extraction of tissues, with determination of boiling points, iodine number, degree of saponification, acrolein reaction and specific color reaction. Similarity of chemical structure will add much valuable information concerning the pathogenesis of lipoid pneumonia in individual cases.[14] Sometimes the lipoid pneumonia presents itself as a "coin lesion",[15] or as in our case 1 as a localized mass paraffinoma.[16] Then its differentiation from bronchogenic carcinoma is difficult, and surgical removal of the lesion will prove the diagnosis. The concomitant occurence of carcinoma of the lung and chronic oil pneumonia has been rarely reported, yet it has been suggested that aspirated mineral oil may occasionally cause lung cancer in man.[15,17,18] Few examples of lipoid pneumonia associated with atypical mycobacterial infection have been reported.[15]

Treatment

When lipoid pneumonia is localized, cure has been affected by surgical resection of the involved area, which pre-operatively may have been thought to have been due to carcinoma or chronic unresolved pneumonia.[19] Symptomatic treatment with antibiotics for infection and a trial of adrenocorticoids may be helpful. Prednisolone has effected improvement in a few instances clinically, radiologically, and in lung biopsy specimens.[10,20] It therefore, merits

TABLE 2: Pulmonary Function Tests (Case 3)

Static Lung Volumes	*Measured*				*Predicted*
	ā bronchodilator		p̄ bronchodilator		
IC	1.32	63%	1.37	65%	2.07 L
ERV	0.51	49%	0.49	46%	1.04 L
VC	1.83	59%	1.86	60%	3.12 L
RV	1.50	91%			1.65 L
FRC	2.01	75%			2.69 L
TLC	3.33	70%			4.77 L
RV/TLC		45%			35%
End tidal CO_2	29mm Hg				

Arterial Blood & Gas Exchange

	Rest		*Exercise*	*100% O2*
DL_{CO}	15	69%	22 ml/min/mm Hg	
Pa_{O_2}	56		47	225
Sa_{O_2}	86		79	100
Pa_{CO_2}	32		36	40
pH	7.42		7.41	7.43
Hemoglobin	10.8			
Sa_{CO}	5.8			

Mechanics of Breathing

	Measured		*Predicted*
% $FEV_{1.0}$	1.23	70%	72–98%
% $FEV_{3.0}$	1.58	90%	92–100%
MVV	55.1	63%	87.3 L/min

* IC = Inspiratory capacity, ERV = Expiratory Reserve Volume, VC = Vital Capacity, RV = Residual Volume, FRC = Functional Residual Capacity, TLC = Total Lung Capacity, RV/TLC = Residual Volume, Total Lung Capacity Ratio. DL_{CO} = Diffusing capacity for carbon monoxide, breathholding method. Pa_{O_2} = arterial oxygen tension, Sa_{O_2} = arterial oxygen saturation, Pa_{CO_2} = arterial carbon dioxide tension, Sa_{CO} = carboxyhemoglobin saturation.

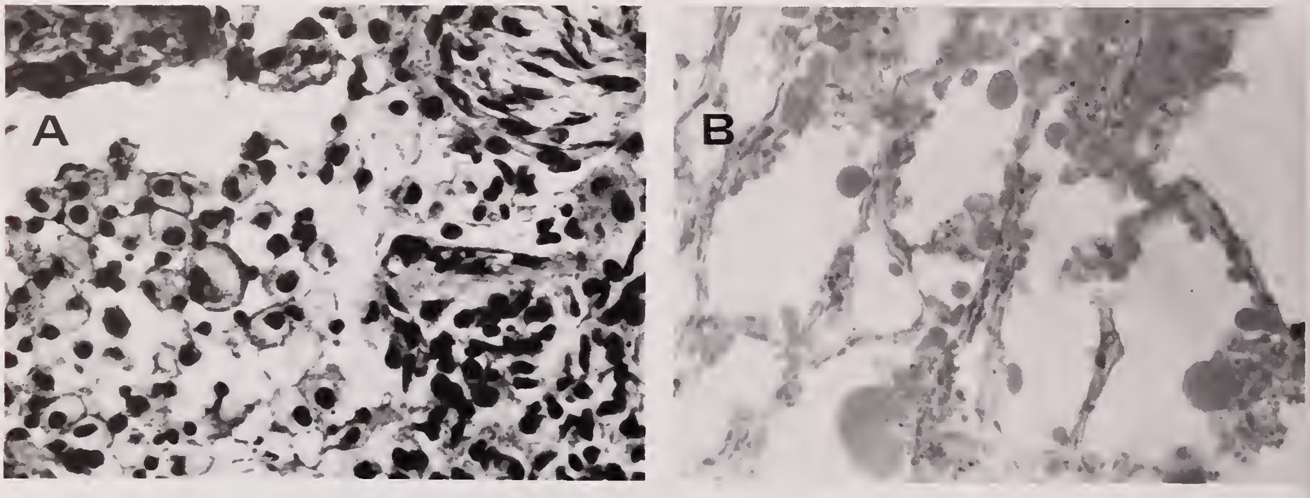

FIG. 5A. Case 3. Alveolus filled with foamy macrophages and occasional lipoidal inclusions. Proliferation of fibroblasts and mononuclear infiltrate in the septum. 5B. Case 3. Lipid droplets in the alveolar spaces.

further trial in diffuse lipoid pneumonia. Isoniazid chemoprophylaxis is indicated when concomitant breakdown of healed tuberculosis is feared.[20] As to whether bronchopulmonary lavage can help, particularly in the early reversible phase, needs further clarification.

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Testicular Feminization (Morris' Syndrome)* Report of a Case

AUGUSTUS O. GODETTE, M.D.† AND JOHN F. J. CLARK, M.D.‡

Report of Case

V.F., a woman 19 years of age, attended the emergency room with a chief complaint of lower abdominal pain. She was found to have primary amenorrhea and was admitted to the hospital. She had been seen by many physicians and had been treated variously for lower abdominal pains. On her last visit to a doctor she was told to have a gynecologic evaluation because she was "close built."

Her maternal aunt, who is married, aged 38, has never menstruated. Her 3 sisters, ages 16, 15, and 12 are judged to be normal. She was well-developed and well-nourished, 5 ft. 7½ inches tall, weighing 132 lbs., with broad shoulders and narrow hips (figure 1). Female fat distribution was conspicuously absent. She had normal breast development. Pubic and axillary hairs were scanty. The labia majora and minora were large and somewhat redundant with some wrinkling of the skin of the labia majora. The clitoris was slightly enlarged, and the vaginal oriface was small, with intact but perforated hymen. Palpation of the inguinal regions revealed 2 2 x 3 cm solid, mildly tender masses which were freely movable, smooth, and seemingly unattached to any structures. The vagina could not accommodate 2 fingers, and the small finger could be inserted only to about 4 cm. Speculum and bimanual examination were abandoned in favor of rectal examination which revealed no palpable pelvic organs.

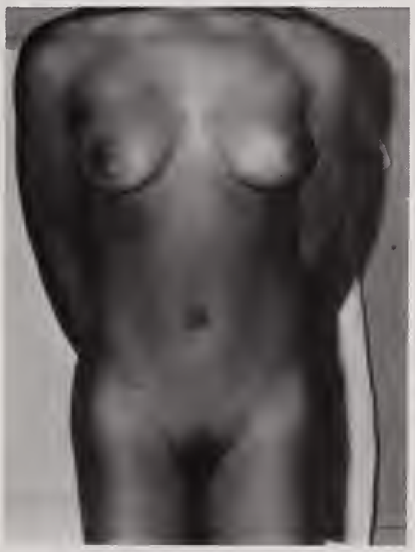

FIG. 1. Note broad shoulders and narrow hips but good breast development. Also, note absence of female fat distribution and pubic hair.

X-ray films of long bones revealed epiphyseal lines in the distal radial bones which ordinarily are completely closed by age 19. This is a very constant feature in normal females. IVP was negative for congenital anomalies. Vaginagram revealed a shallow vagina, and only a small balloon was seen to fill with contrast medium. The depth measured approximately 3 cm. Pelvimetry showed a classically android pelvis.

Impression: Possible testicular feminization syndrome and gonadal dysgenesis.
Routine laboratory examinations were within normal limits.

		Normal Age 20	
		Male	Female
Urine for pituitary gonadotrophic hormones	0 Int. U		
Urine for 17 keto steroids	15.8 mg/24 hrs.	6–21	4–16
Urine for 17 hydroxy steroids	3 mg/24 hrs.	2–7	lower

Buccal smear – chromatin-negative
Peripheral blood-no definite female 'drumsticks' in 500 neutrophils.

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A laparotomy was performed, and the gonads were removed through a Pfannenstiel's incision from the inguinal canals along with the infundibuliform fascia by transecting the spermatic cord which extended from the testes toward the respective kidneys (figure 2). The pelvic cavity was found to be free of pelvic organs. Figure 3 shows the histological appearance of the gonads.

The patient was placed on estrogen maintenance and is doing well. We are prepared to perform vaginoplasty if and when we deem it necessary. During her follow-up in our clinic the vagina was dilated bimonthly over a period of about 4 months, with graduated test tubes at each successive visit. This resulted in significant enlargement of the vagina, enough to accommodate a test tube about 2.5 cm in diameter and 10 cm in length. The dilatation was accomplished without significant discomfort.

After several weeks of sexual contact there is no dyspareunia, and the vagina now admits an adult medium-sized speculum without pain.

Discussion of Testicular Feminization (Morris' Syndrome)

Although individual cases had previously been described by several authors, Morris (1953) first clearly defined the syndrome. Affected individuals are male (XY) pseudohermaphrodites and are usually tall (average, 5 ft., 8 inches), good-looking, intelligent, and attractive female phenotypes. At puberty, which is often later than normal, the breasts develop normally, and the external genitalia are female, but the vagina may be short and always ends blindly. There is no uterus or Fallopian tubes. There is, of course, primary amenorrhea, although the phenotypic heterosexual drive is usually well-developed. Pubic and axillary hair tend to be sparse and may be virtually absent.

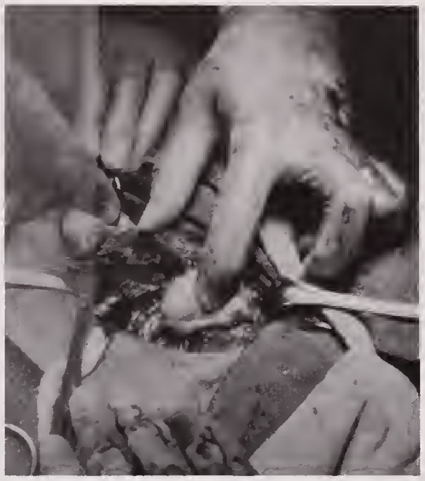

FIG. 2. Bilateral testes. Note well-developed tunica vaginalis under the infundibuliform fascia and a well-developed spermatic cord.

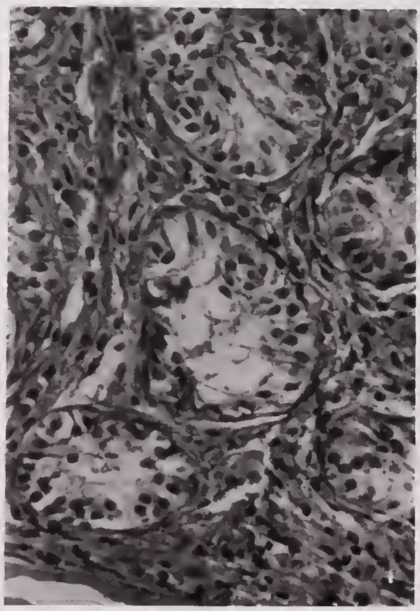

FIG. 3. Histological examination of gonads revealing seminiferous tubules, infantile in size, shape and configuration. There is a slight increase in connective tissue surrounding them and also an increase in basement membrane. There is also a relative increase in the interstitial cells of Leydig which indicates a high pituitary gonadotropin secretion. There is absence of spermatogenesis and relative lack of Sertoli cells.

The sex chromosome constitution is XY, and the gonads are testes. In 19% of cases the testes are situated in the labia majora, in 60% in the inguinal canal, and in 21% within the abdomen, usually on the side wall of the pelvis. In postpubertal patients the testes show Leydig-cell hyperplasia, and the seminiferous tubules are of small caliber, may have no lumen, and are lined by indifferent epithelial cells. Spermatogenesis is absent (Hauser, 1963;[1] Simmer et al, 1965[3]).

Etiology and mechanism. The output of estrogens by these individuals is higher than in men but slightly lower than in average normal women. Vaginal smears support this. The urinary output of 17 ketosteroids is about the same as in normal men, or occasionally higher. Pituitary gonadotropin levels are somewhat elevated in some, normal in others (Hauser).[1]

The investigations of French et al[2] (1966) on 2 patients strongly suggest that the defect in this syndrome is a lack of target organ response to testosterone. Both plasma and urinary levels of testosterone are similar to those of normal adult males, and there is a lack of the usual anabolic effect of large doses of testosterone propionate.

It appears that because of this insensitivity to testosterone, pituitary gonadotropin secretion is increased (no feedback effect; apparently the hypothalamus must also be insensitive). This accounts for the Leydig-cell hyperplasia of the testes and for the estrogen secretion. The estrogens are formed by the testes in amounts sufficient to produce almost normal female blood and urine levels, and to induce breast development. This estrogen secretion prevents excessive secretion of pituitary gonadotropins (predominantly LH), and is sufficient to produce feminine development. The above average height probably reflects insensitivity of the epiphyses to testosterone, and it is not until estrogen secretion rises (apparently slowly) that growth ceases.

As to the scantiness of axillary and pubic hair (35% have no pubic hair, in 40% it is scanty, and in 15% there is a normal escutcheon; axillary hair is more frequently absent than is pubic hair). This may reflect insensitivity of the hair follicles to testosterone, as postulated by Wilkins and Prader[4] in 1957. Seemingly, the growth of sexual hair requires the synergistic effect of both androgens and estrogens.

As to the genetic mechanism involved, a strong familial history has been noted since the turn of the century. The condition is passed on from apparently healthy mothers to genotypic (XY) males. In families in which more than 1 member is affected the overall ratio of intersex offspring to normal male siblings is about 3:1.[2] A recessive gene governing the insensitivity to testosterone seems to be almost certainly involved. This could be on an autosome but is more likely to be sex-linked, i.e., on an X chromosome. Axillary and pubic hair may be sparse in the mothers of these patients.

Frequency. Prader estimated the incidence of testicular feminization to be 1 in 20,000; Hauser believes it to be nearer to 1 in 2,000.

Diagnosis. This is rarely made before puberty unless an older sibling is affected or unless a "lump" is noted in the labia or inguinal region of a young girl. Buccal smears are chromatin-negative. After puberty the absence of sex chromatin in buccal smears should give the diagnosis in a phenotypic female with primary amenorrhea and a short, blind vagina. Tumors (mainly seminomas) may develop in the abdominal testes and, as in cryptorchidism, are about 10 times more frequent than in normal males.

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Hoarseness and Dysphagia Secondary to Cervical Hyperostosis*
Report of an Unusual Case

HARRY C. PRESS, JR., M.D.† AND LASALLE D. LEFFALL, M.D.‡

Hyperostatic spurring of the cervical vertebrae may occur without clinical symptoms. However, when spur formation of the anterior vertebral bodies is marked, involvement of the larynx, pharynx or esophagus may occur.

Symptoms previously reported include hoarseness[3], dysphagia[2,4], Horner's syndrome[5], and "lump" in the throat[6]. Our patient had similar symptoms, but his case is unusual in that the magnitude of spur formation is massive when compared with previously reported cases.

Report of Case

A 58-year-old man complained of hoarseness and a "change of voice" for 2 weeks prior to seeing his physician. A history of an occasional "lump" in the throat associated with mild dysphagia and throat irritation was obtained, antedating present symptoms by about 6 months. Periodic substernal pain had occurred, unassociated with the dysphagia or deglutition. The man was admitted to our hospital for a work-up for possible tumor.

In recent years the patient has been treated for arthritis which included symptoms of a "stiff neck." Also noted was the patient's occupational history of prize-fighting which terminated some 25 years ago. He has since worked as a railroad parlor car attendant. The remainder of the history was non-contributory.

Physical examination revealed a well-developed, well-nourished male in no acute distress. The voice was moderately hoarse. Two palpable nontender lymph nodes were found in the right posterior cervical region. The neck was somewhat rigid but not painful on flexion or extension. The tonsillar pillars were hyperemic, with mild injection of the oral pharynx. Other physical findings were essentially negative.

Roentgen Findings

Roentgen examination of the cervical spine revealed marked hyperostatic spurring of the anterior vertebral bodies. X-ray films (Figures 1 and 2) demonstrated the following:

1. Good alignment of the cervical vertebral column.
2. No alteration in vertebral body height.
3. Maintenance of disc interspaces.
4. Bridging of the hypertrophic spurs along the bodies of C-2 through C-7.

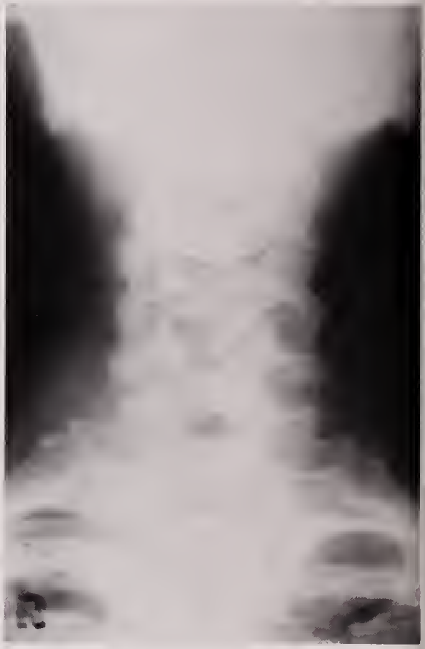

FIG. 1. Anteroposterior view of cervical spine showing severe hypertrophic spur formation along bodies of C_2 through C_7.

5. Spur formation most marked on the left.

Contrast radiography of the pharynx and esophagus using barium revealed (figures 3 and 4):

1. Marked compression of the posterior pharyngeal wall and cervical esophagus from C-3 to T-1, more marked on the left.
2. Indentation of the posterior pharyngeal and esophageal mucosa secondary to pressure from the cervical spurs.

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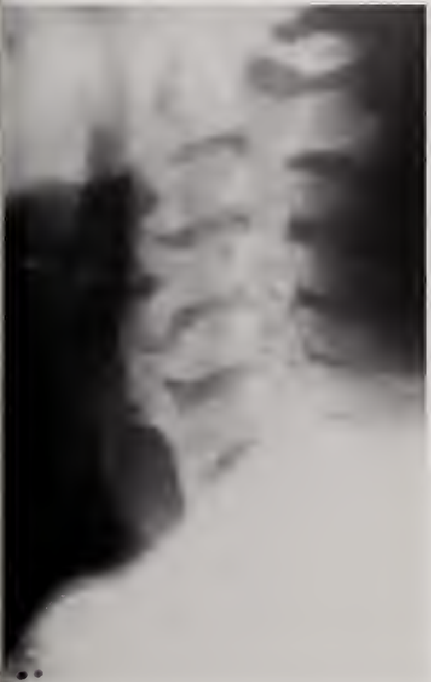

FIG. 2. Lateral view of cervical spine showing severe hypertrophic spur formation along bodies of C_2 through C_7.

3. Pressure on the esophagus with deviation to the left.

Further work-up included direct larygoscopy and esophagoscopy under local anesthesia. Edema and hyperemia of the left pyniform sinus were noted. The epiglottis, vocal cords, arytenoids, and right pyniform sinuses were normal. No definite tumor mass was seen in the pharynx or esophagus. Biopsy of the left pyniform sinus was done in 2 areas. A pathological diagnosis was made of chronic nonspecific inflammation of the pyniform sinus.

To further explore the possibility of a tumor mass, surgical exploration of the neck was done 1 week later. An incision was made into the neck along the anterior border of the sternocleidomastoid muscle. Elevation of this muscle revealed a large internal jugular lymph node, part of which was sent for frozen section. "Reactive" hyperplasia of lymph nodal tissue was reported. Further exploration of the neck in the region of the cervical spine showed areas of overgrowth of the vertebral bodies which had spur formation. No tumor was located in the area of the esophagus or left sternocleidomastoid area. No attempt at surgical removal was made. The procedure was tolerated well with rapid recovery, and the patient was discharged to be followed on an outpatient basis.

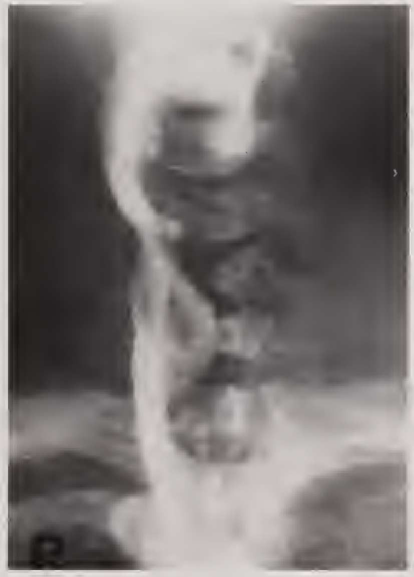

FIG. 3. Anteroposterior view of cervical area with barium revealing non-filling of left side of pharynx and cervical esophagus due to extensive pressure by hypertrophic spurs.

No recurrence of symptoms has been noted after 18 months. Hoarseness and pharyngitis subsided upon discharge, with improvement of dysphagia and the globus sensation. Should symptoms recur, surgical removal of the cervical spur is planned.

Discussion

This case demonstrates hyperostosis of the cervical vertebrae producing laryngeal and esophageal symptoms. These symptoms are secondary to pressure effects upon the larynx, pharynx, esophagus, and/or cervical nerve roots.

The mechanism of laryngeal symptoms has been explored by Gribovsky.[2] According to his theory, pressure from the lateral spur projection may involve the recurrent laryngeal nerve with subsequent hoarseness. Similar pressure effects upon the pharynx and esophagus are believed to produce dysphagia and the globus sensation. Since no therapeutic surgical procedure was attempted, improvement of symptoms may have resulted from a shift of pressure effects during surgical manipulation.

The prize-fighting years before may have accelerated the cervical hypertrophic spurring.

Bick[1] believes "pathogenically" spurs are characteristic reactions to strain found at the insertions of powerful ligaments into the bone at many sites throughout the axial and appendicular skeleton.

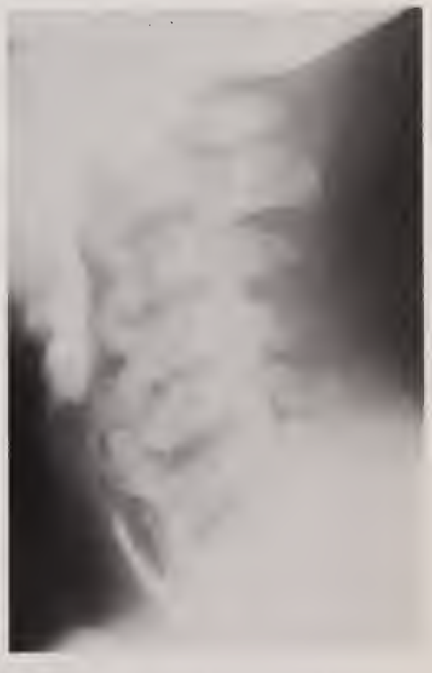

FIG. 4. Lateral view of cervical area with barium revealing compression of posterior pharyngeal wall and cervical esophagus by hypertrophic spurs.

There has been a paucity of cases reported in the literature involving symptomatic cervical hyperostosis. Heck[3] presented 4 cases associated with hoarseness and related symptoms in 1956. Helding[4] searched the literature in 1960, only to disclose a total of 36 cases associated with dysphagia. Horner's syndrome has also been reported.[5]

The case presented, involving hoarseness, dysphagia, globus sensation and pharyngitis, only emphasizes the fact that hypertrophic spurs of the cervical spine should be included in the differential diagnosis of such symptoms. The fact that it has been rarely reported does not necessarily mean it is a rare condition. There could well be a lack of recognition of cervical spurs as an etiological possibility of such symptoms, and a diagnosis of "globus syndrome" made.

Roentgen examination should include plain films of the cervical vertebrae as well as contrast radiography of the pharynx and cervical esophagus. Routine esophagrams without examination of the cervical area are inadequate in patients having the above symptoms.

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The Diagnostic Evaluation of the Patient With Hypoglycemic Symptoms

JAY R. SHAPIRO, M.D.* AND FARANAK SOTOUDEH, M.D.†

Each month the Medical Annals will publish a discussion of a topic involving clinical management particularly as related to office practice. These will be brief and will include general references. The Continuing Education Committee would appreciate comments from readers as to future topics, or the usefulness of the articles to the practitioner.

Either through organizational efforts or by word of mouth, the public is aware of the non-specific symptoms that may be caused by lowered blood glucose. Also, it appears that many persons are convinced, at least temporarily, that frequent injections of adrenocortical extract or rigid adherance to low carbohydrate diets will relieve these vague symptoms. The physician faced with the patient who attributes symptoms to low blood sugar has the following to consider initially: does the blood glucose fall rapidly enouth, or to levels low enough to cause symptoms; is there a functional or organic basis for the low blood glucose; can the patient be weaned from the multiple medicines usually prescribed previously before testing is carried out? Significant symptoms of hypoglycemia generally occur when blood glucose is less than 50 mg/100 cc. Hypoglycemia of *functional* cause (early diabetes mellitus, subtotal gastrectomy, thyrotoxicosis, and reactive or idiopathic hypoglycemia) is not characterized by low fasting blood glucose. Patients with severe hepatic disease, Addisons disease, or hypopituitarism may or may not have fasting hypoglycemia. Patients found to have hypoglycemia after a 12 hour fast, particularly those not known to have recently consumed large quantities of ethanol, must be considered as having hypoglycemia of *organic* etiology. Insulinoma, islet cell hyperplasia, large retroperitoneal tumors (fibrosarcoma) must be considered as diagnostic possibilities. Hypoglycemia due to insulin or sulfonylureas may be unsuspected in the comatose psychiatrically disturbed patient. Several other drugs may uncommonly cause hypoglycemia: propranolol, phenylbutazone, tripelennamine, salicylates, iproniazide, dextropropoxyphene, and oxytetracycline are among those previously cited.

Where fasting hypoglycemia is documented the following procedures should be carried out. Hospitalization may be necessary for some, but not all patients where this protocol is followed.

1) Five hour glucose tolerance test: Bloods should be drawn at one-half hourly intervals in order that brief nadir not be missed. Insulin determinations should be done on specimens with values less than 50 mg %. Patients with reactive hypoglycemia have low values at 2 hours, while early diabetics, subjects with liver disease, hypopituitarism, and Addisons Disease have low values at 3-5 hours. A normal or diabetic curve may occur in patients with insulinoma. The patient with malabsorption will have a flat curve. One must insure that the patient has had adequate CHO intake for 3 days prior to the test, particularly in patients purposely restricting CHO.

2) IV Tolbutamide Tolerance Test (IV TTT): An IV drip of saline should be kept open during the test, and

TABLE I: The Symptoms of Hypoglycemia

I The Autonomic Phase	II Cerebral Phase	III Brain Stem Phase
restlessness	slow mentation	progressive coma
tremor	irritability	hypotension
palpitations	agressiveness	death
sweating	negativism	
hunger	bizarre behavior	
rise in BP	lethargy	
tachy- or bradycardia	confusion	
	somnolence	
	convulsions	

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glucose held ready to administer. Following one gram of tolbutamide, blood should be drawn for glucose and insulin at 15″, 30″, 60″, 90″, 120″, and 240″. A fall in blood glucose to 60% of baseline by 30″, elevation in plasma insulin to over 150 uU/ml, and delayed (over 2 hours) return of glucose to baseline levels all characterize the positive test. Patients with liver disease, hypopituitarism, and malnutrition may have a false positive test, but levels generally return to normal by one hour. False negative IV TTT do occur, and in this case, elevated plasma insulin values may indicate the presence of abnormal pancreatic responsiveness. A less than adequate fall in blood glucose will be seen in the diabetic. A normal test is seen in patients with retroperitoneal tumors.

3) Leucine Tolerance Test: Amino acids such as leucine or argenine both stimulate insulin release, and diminish hepatic gluconeogenesis. Following the administration of 150 mg/kg leucine, either IV or orally, bloods are drawn every 10 minutes for 90 minutes (for glucose and insulin). A positive test is noted in 50% of patients with insulinoma. A false positive test may occur in patients recently treated with sulfonylureas. This test is of value in the hypoglycemic child suspected of having leucine sensitivity.

4) The 48–72 Hour Fast: This is probably the most potent test, and one that requires hospitalization. Either symptomatic hypoglycemia, or evaluation of plasma insulin out of proportion of blood glucose will be seen by 48 hours in 70% of those patients with organic hypoglycemia. Blood glucose should be determined every 4-6 hours throughout the day and night, and the ward personnel alerted to the procedure.

A test recently proposed for the diagnosis of insulinoma is the measurement of proinsulin in blood either fasting or following IV tolbutamide. While this appears to have promise, it is in the developmental stage, and the determinations are not readily available.

In terms of diagnostic usefulness, the above procedures may be listed in the following order: 48-hour fast, IV tolbutamide, leucine tolerance, and the 5-hour GTT. Adjunctive procedures include x-ray studies of the sella turcica and thyroid and adrenal function tests. Retroperitoneal tumors associated with hypoglycemia are usually large and may be detected by conventional radiographic studies. Pancreatic angiography may assist in uncovering pancreatic neoplasms, although many will probably escape detection because of their small size.

The differential diagnosis in the symptomatic patient includes pheochromocytoma, orthostatic hypotension the hyperventilation syndrome. Additional rare causes of hypoglycemia include adrenal carcinoma, the glycogen storage diseases, hereditary fructose intolerance, idiopathic familial hypoglycemia. The possibility of co-existing adenomata in parathyroid glands, pituitary, or adrenal should be considered in the patient with organic hypoglycemia due to islet cell adenoma.

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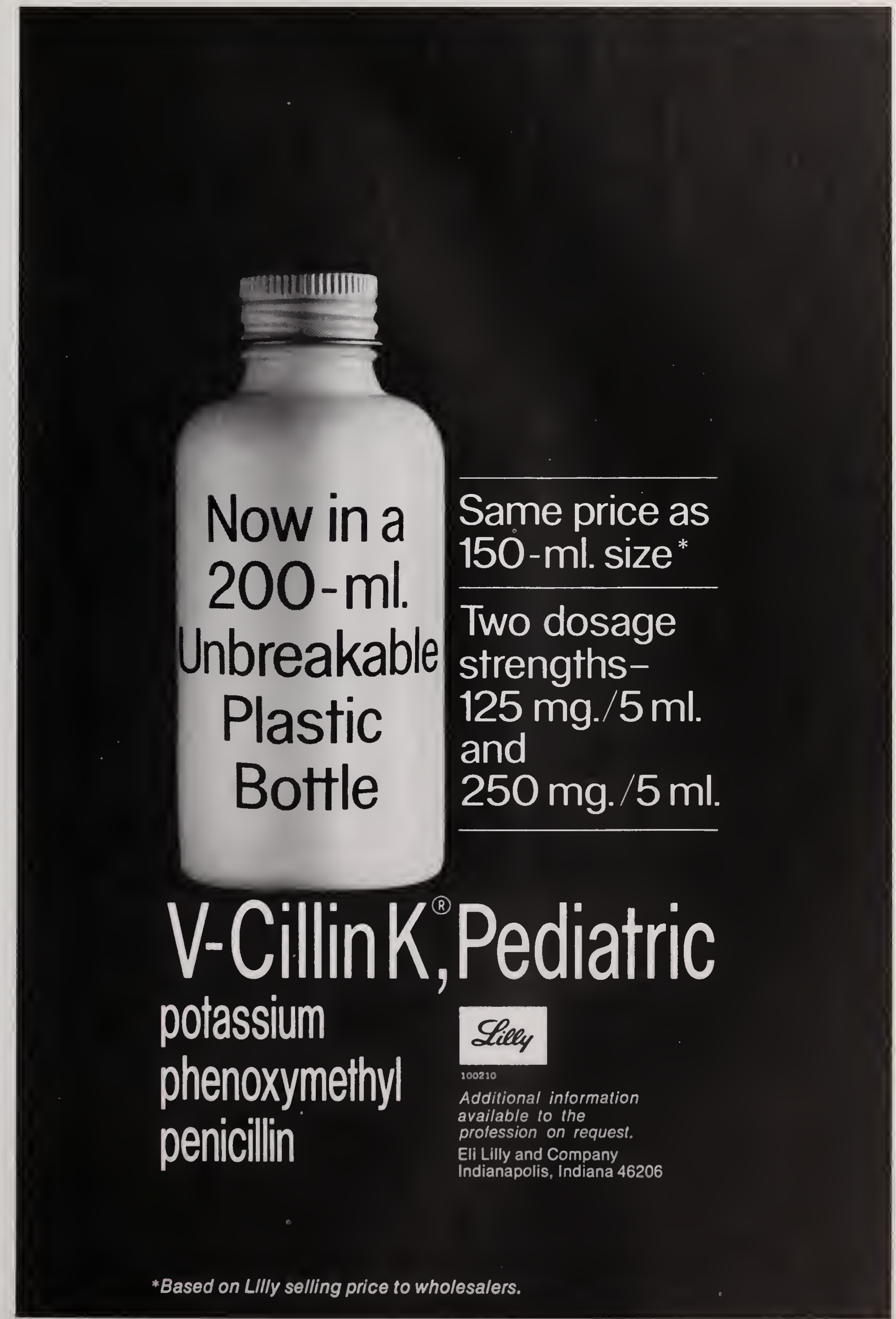

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1. Demeulenaere, L.: Action du R 1132 sur le transit gastro-intestinal, Acta gastroent. Belg. 21:674-680 (Sept.-Oct.) 1958.

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Progress in Community Health Services

Promoting good health and preventing disease and disability of the residents of the District of Columbia and of visitors to the Nation's capital are the principal objectives of the Community Health Services Administration of the Department of Human Resources. CHSA serves as a municipal and state health agency, the only one of its kind in the nation.

Carrying out these objectives, the Administration plans, implements and directs many public health programs, including the enforcement of applicable laws and regulations which effectively maintain and improve the health and well-being of the people of the District of Columbia.

During last year's reorganization of the Administration, CHSA altered its organizational units and further implemented its neighborhood health care concept. Neighborhood Health Centers, now operating under the Directorate of Clinical Services, provide primary medical care for sick patients of all ages and also preventive health services, including immunizations, routine health examinations and health education.

Consumer services provided by the Community Health Services Administration, briefly summarized, include:

* Offers a variety of health educational, medical, screening, diagnostic, consultant, preventive, treatment and rehabilitative services.

* Provides the leadership for licensing health facilities in the District of Columbia, including Nursing Homes and Personal Care Homes.

* Initiates and coordinates contractual arrangements with voluntary agencies for the provision of community health services.

Accomplishments in a number of our major programs of our past fiscal year are worthy of note. For example, under our Maternal Health program, 4,379 patients were served and 18,018 clinic visits made. During the first year (3 months) of operation of the Birth Control program in 1964, 569 patients were served and 679 clinic visits made. During the last fiscal year 17,829 patients were seen and 34, 597 clinic visits made.

Effectiveness of our Immunization Program is reflected in the following disease categories—diphtheria, no cases since 1966; polio, one case in 1968; smallpox, no cases since early 1930's; rabies in man, no cases; and rabies in animals, no cases since 1968. During the past year mass immunization campaigns against regular measles and rubella and polio were conducted in schools and our clinics.

The Supplemental Foods Program was started in June, 1969, to provide extra nutritional food for eligible expectant and postpartum mothers, infants and children through age 5 years. During the first year of operation, fiscal 1970, 84,705 bags of food were distributed. In fiscal year 1971, the figure had almost doubled to 165,000 bags of supplemental foods.

In the area of extended care facilities, we have successfully prosecuted the operators of 16 personal care homes and the administrators of 5 nursing homes for repeated violations through the United States District Court, D.C. Superior Court and the D.C. Board of Appeals and Review. Our primary concern is for quality care and services in facilities meeting the optimum standards for all patients and residents of health care facilities in the District of Columbia.

During the past year, 3,376 children have been screened for the detection of lead poisoning through a program in the Neighborhood Health Centers and a special program funded by the Model Cities Commission. Twenty per cent of the children screened were found to have elevated blood lead levels. These children were referred for additional diagnosis and treatment.

In the Neighborhood Health Centers during the calendar year 1970, physician service visits increased by 32,955. In 1970, there were 100,317 physician service visits as compared with 67,362 visits in 1969. Of the total visits, 55% were in the area of pediatrics, 27% adult medicine, and the remaining 18%, obstetrics and gynecology.

Plans are underway to establish clinic-type pharmacies in the most strategic locations in the District. The prescription workload went from 329,159 in 1968, to 524,268 in 1969, and rose to a high of 530,693 in 1971. In 1961, the workload was only 197,243.

Newly diagnosed tuberculosis cases dropped from 380 in 1969 to 370 in 1970. New cases of infectious syphilis have been fluctuating in an erratic way over the last decade. For example, there were 472 cases in 1960, 702 in 1962, 468 in 1966, 772 in 1968, 539 in 1969, and 515 in 1970. There were 13,947 reported cases of gonorrhea in 1969 and 13,500 cases in 1970. Ninety-five per cent of the reported cases, however, were obtained from the public clinics, and only 5% from private physicians.

Health care was provided to many hundreds of people by the Community Health Services Administration during 30 assemblies, demonstrations, inaugurals, and a riot. From 1965 to June 1971, about 57,775 persons were trained in Medical Care Self-Help.

A Pediatric Nurse Associate (Practitioner) program was inaugurated in 1971 to more effectively utilize the particular skills of the nurse under the physician's supervision. Health services for infants and children and the management of the total patient are provided by these highly skilled professional nurses.

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MEDICO-LEGAL REPORT

Medico-legal matters are becoming an increasing consideration in the daily practice of medicine. Considerable material in this area appears regularly in both the professional and general press. For these reasons we feel it would be of interest to the members of the Medical Society to have articles appear in a monthly column pertaining to the legal aspects of medicine—with particular emphasis on problems as they develop in the Washington Metropolitan area. While malpractice items may be in many ways the most dramatic cause of concern, numerous other areas such as office zoning, licensing, acting as an expert witness, medical testimony, dying declarations to a physician, insurance items, public policy, authorization by the patient for clinical investigation and hospital liability are also areas in which the physician needs to be knowledgeable.

Among professional liability problems such as informed consent, treatment without consent, improper commitment, drug reactions, erroneous diagnosis and what constitutes abandonment as well as improper disclosure, and invasion of the patient's right to privacy, are all areas in which the interpretation of the law has been changing.

The view growing in American law that every injured person should be entitled to compensation may result in medical malpractice being covered by a no-fault system, such as that already existing in some states in the area of automobile accidents. Another system being considered is that of compulsory or binding arbitration.

We will endeavor to cover these and other items in future columns to be written by both knowledgeable colleagues and guest writers specializing in other appropriate fields.

HARVEY AMMERMAN, M.D.
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DARRELL C. CRAIN, M.D.

President, The Medical Society of the District of Columbia, 1972

Darrell C. Crain, M.D., F.A.C.P.

The 105th President of the Medical Society of the District of Columbia, Dr. Darrell C. Crain, assumed his new office on January 1, 1972.

Dr. Crain, a native of Washington, D.C., was born March 29, 1910, and received his M.D. degree from George Washington University in 1932. He took his postgraduate hospital training at the Central Dispensary and Emergency Hospital in Washington, D.C., where he spent one year in a rotating internship and one year as Assistant Resident in Medicine. At Walter Reed Army Hospital he was Rotating Medical Officer 1934-1935, Associate Medical Officer, Arthritis Ward, 1935-1936, and Chief Medical Officer, Arthritis Ward, 1936-1937. He began private practice in 1937, which he left during the War years 1942-1945 when he held the commission of Major in the United States Army Medical Corps. In 1945 Dr. Crain resumed private practice which he has maintained to the present. His practice in Internal Medicine at 1234-19th St., N.W. is limited to diagnosis and treatment of arthritis and rheumatic diseases.

He is currently the Director of the Arthritis Rehabilitation Center in Washington, D.C. and a member of the teaching staff at Georgetown University School of Medicine as Clinical Professor of Medicine.

From 1947 to 1969 he served as Consultant to the Surgeon General of the United States Public Health Service at the Bethesda Clinical Center. He is presently the Associate Head, Department of Medicine and Chief, Division of Rheumatic Diseases at Doctors Hospital, Director of the Rheumatology Clinic at Georgetown University Hospital, and Consultant to the Surgeon General of the United States Army in Arthritis and Rheumatic Diseases at Walter Reed Army Hospital.

Dr. Crain has been active in the affairs of the Medical Society of the District of Columbia since his election to Active membership in 1938, including terms as chairman of the Program Committee and the Cooperation with the District of Columbia Bar Committee as well as membership on the Regional Medical Program Committee. He served as First Vice-President in 1965 and was President-Elect for 1971.

The Local Chapter of the American Rheumatism Association (the District of Columbia Rheumatism Society) was organized in Dr. Crain's home in 1946. He is also a co-founder, in 1947, of the Local Chapter of the Arthritis foundation, now known as the Arthritis and Rheumatism Association of Metropolitan Washington. In addition to his membership in the Medical Society of the District of Columbia, Dr. Crain is affiliated with several other professional societies, including the American Medical Association, The American Society of Internal Medicine, the American Society of Clinical Pharmacology and Therapeutics, the American Congress of Physical Medicine and Rehabilitation, the Southern Medical Association, the New York Academy of Sciences, the Pan American Medical Society, the International Medical Society, the American Association for the Advancement of Science, and the Washington Academy of Medicine. He is also a Fellow in the American College of Physicians and a Diplomate of the American Board of Internal Medicine.

He serves on the Medical Advisory Board for the Visiting Nurses Society of Washington, D.C. and is a member of the Board of Directors of the Westminster Foundation at Annapolis, Maryland. Dr. Crain is also a member of the Board of Directors of the Arthritis and Rheumatism Association of Metropolitan Washington, a position which he has held since 1948.

Not the least of Dr. Crain's accomplishments has been the publication throughout his career of numerous scientific works, the most recent of which is "The Arthritis Handbook; A Patient's Manual on Arthritis, Rheumatism and Gout," published in 1971 by Exposition Press. He has also been a frequent contributor to the MEDICAL ANNALS.

Dr. Crain married Leslye Louise Moore of Washington, D.C. and Richmond, Virginia, in 1934. The Crains, who reside at 6422 Garnett Avenue, Chevy Chase, Maryland have a son, Darrell C. Crain, III, and two daughters, Mrs. Barbara Rollinson and Mrs. Ann Fitzgerald, as well as six grandchildren.

In his leisure time Dr. Crain is a collector of medals. He possesses the only complete collection of presidential inaugural medals outside of the Smithsonian Institution, an interest perhaps inherited from his father, who was the Washington jeweler and artist who designed the inaugural medals for presidents Harding, Wilson and Coolidge.

A dynamic and eloquent man, Dr. Crain promises to be a most effective President. The Medical Society is fortunate in having for its new president a person of Dr. Crain's energies.

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d.c. MENTAL HEALTH ADMINISTRATION

Halfway Houses

George was telling the group, "Well, there I was, out of a job, nothing to do but stare at those four walls and getting mighty fed up so, in came a couple of guys from upstairs with a bottle . . . 'Have a drink with us' . . . I said 'no thanks, I'm off the stuff' . . . 'Oh, come on, George one little drink ain't gonna hurt you' . . . I knew better–but there I was off again on another spree." This is a tale heard over and over.

The Unmet Need

Eighty-five to 90% of the city's estimated 100,000 alcoholics are still functioning in the community. They are working when not on a spree and when jobs are available. Many still live with their families, but a substantial number (three-fourths of those applying for outpatient treatment) are separated, divorced, widowed or single. Drinking frequently has alienated these men from former friends as well and their current social ties are most tenuous as they move from rooming house to rooming house. Many of them have tried to do something about their alcoholism but have lost hope cooped up in some dingy room, uninvolved with people.

After a drinking bout, they, in the past, would often get arrested and sent to the Workhouse. Now they are likely to land in a Mental Health Administration Inpatient facility. Upon release from either place they are usually faced again with the same dreary living conditions.

Actually, extensive hospitalization most often is *not* the answer to these persons needs, but outpatient therapy once a week is not sufficient either.

An understanding of this situation caused a perennial demand for Halfway Houses by alcoholism clinics, conferences, committees, community organizations and by the Hagan Bill (Alcoholic Rehabilitation Act of 1967–P.L. 90-452).

Attempts to Meet the Need

Several private Halfway Houses, usually under the sponsorship of Alcoholic Anonymous members, had opened and closed again in the District of Columbia in the dozen years preceding 1967.

Funding for this type of facility was extremely slow in forthcoming. The first public Halfway House in D.C. was opened by the Department of Vocational Rehabilitation with grant money in 1967–a 10-bed facility for alcoholics selected by DVR counselors at the Rehabilitation Center for Alcoholics.

In August of 1968, in response to the Health Department's budget request, Congress appropriated $100,000 for fiscal year 1969 to the Health Department to contract for halfway house beds for alcoholic patients in order to provide them "with a place to live thereby making it possible to continue treatment in the outpatient clinic facilities of the Department." A simple mathematical error in hurried calculations during congressional hearings led to a plan to make available 138 beds at the rate of $2.00 per day per person. That such a plan was hardly feasible soon became evident. The only organizations willing to provide this service for $2.00 were those which had already been providing free beds to the destitute: the missions. Contracts actually were drawn up with 2 of these organizations, but in vain. A report on the status of the Halfway House contracts, dated September 1969, stated: " 'down-and-out' patients, not interested in changing their life style or in treatment, are able to use the existing missions without Health Department assistance. Patients who are desirous of making a change are most anxious to avoid the mission environment. They find it very much conducive to continued or resumed drinking, particularly due to the other men with whom they associate at the missions and the neighborhoods in which the missions are located."

Several other proposals for contracts with interested private groups for developing Halfway Houses came to nought because of insufficient private financial supplements and internal difficulties of the private groups.

Only one contract was successful in setting up a Halfway House with the desired, more closely supervised and therapeutic atmosphere. This contract was with two interested businessmen, who agreed to furnish lodging, food and the salaries for a manager and part-time cook for $3.00 per day per man, supplemented by another $3.00 paid by each man out of his income. Under this contract, the Department's alcoholism center in Area B furnishes the treatment for the residents and has over-all jurisdiction over the development and supervision of the residential facility, including the choice of manager and other staff, house rules and selection of the residents.

The facility started somewhat slowly in a small place. By September 1969 it had housed 22 patients and was in need of expansion. It was moved to a larger house with a 20-bed capacity in November 1969. In June 1970 under the same contractual arrangements another 18-bed house was opened nearby. At this writing, the Mental Health Administration is about to expand again to accomodate both males and females in one of the houses. The women will live in a separate section and have their own female managers. This latest expansion focuses on one of the unmet needs of alcoholic women.

At its inception this type of arrangement was unique in the nation featuring close tie-in with an existing outpatient program for alcoholics and contractual arrangement for room, board and house management.

A different kind of community residential facility was added in January 1970 in a condemned building, renovated by patients from the inpatient Rehabilitation Center for Alcoholics (RCA) which is now a satellite of RCA and offers up to 50 beds to RCA patients.

What is Offered in a Halfway House

Today, George can go into one of the Halfway Houses, where he will share a simple but attractive room with one or two other men. Morning and evening meals are taken together by the residents, lunches can be packed. Recreational activities are available. Monday nights there is a house meeting of all residents, Fridays, an A.A. meeting.

Until he gets employed, The Alcoholism Center's liaison worker will help him obtain temporary public assistance and vocational counseling, and he will be expected to participate in Day Treatment at the Alcoholism Center at least twice a week. A required weekly therapy session will continue after he gets a job. He will contribute to his stay at the Halfway House out of his public assistance or his earnings.

The term Halfway House means a facility half way between an institution and the community. We use the word more specifically for the smaller residential home which provides a supportive, therapeutic atmosphere, and in the case of the alcoholic encourages sobriety. Its goals are to help the individual become psychologically, physically and financially able to re-establsih himself independently in the community. It is neither a transient emergency shelter nor a permanent abode. Length of residence is limited though flexible. The average stay has been approximately 3 months. Each resident's use of the House and possible need to continue in it is reviewed at least every 3 months.

A patient is not permitted to stay in the House if he drinks or if his behavior is too disruptive for the rest of the residents. He must be capable of becoming self-supporting. Following through with treatment at the outpatient center is also required. When the diagnostic team feels that a patient will be unable to meet these requirements, he is not admitted to the Halfway House. If after admission to it a patient starts to drink, he is automatically suspended from the House. He is referred to the Detoxification Center and may be escorted there by the manager. Any decision regarding possible re-admission to the House after such an occurence is based on a joint evaluation by Clinic staff and Halfway House manager.

The success of these types of Halfway Houses is in the process of a thorough study. However, data on the first year of operation showed that 38 of 68 admissions to the House were still in treatment at the end of that year and making an adequate adjustment. Considering that 80 per cent of these residents came from institutions (71% from RCA), the results are gratifying. The 20% who did not come from institutions consisted of 9% referred from the Detoxification Center and 11% previously living in the community. For this group of patients, residence in the Halfway House allowed them to remain in the community instead of having to be sent to an institution. Of the patients admitted in the first year, 97% were unemployed when they entered the House. Omitting those who stayed only a few days, 63% found or were helped to find work or training within the first month of their stay.

Total number admitted by November 1971 was 300.

Problems and Continuing Needs

The currently available beds show that progress is being made in closing a major gap in the continuity of care for alcoholics, but the number of beds is very far from approaching the 600 mentioned in the Hagan Bill.

Many are the patients who are not eligible for the now existing residences. The desirability of a wider range of facilities, meeting different needs is recognized: one of these might be a hostel-like facility for patients who need only a few days lodging until they can get themselves re-established. At the other end of the spectrum, a more permanent residence appears essential for alcoholics who no longer need institutional care but are too disabled mentally, physically or emotionally to successfully adjust on their own in the community.

The Mental Health Administration is exploring ways of funding additional community residences for alcoholics.

ESSEX C. NOEL III, M.D.
Director, D.C. Mental Health Administration

Referrals to Halfway House by Physicians

Can a physician in the community refer patients to the Halfway Houses? Not directly, but he can refer them to the Adams Mill Alcoholism Center or if more appropriate, the inpatient facilities, with the recommendation that they be considered for admission to a Halfway House.

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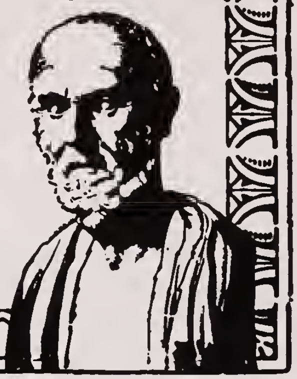

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book REVIEWS

Is the Grass Greener? DOROTHY V. WHIPPLE, M.D. Price, $5.95. Pp. 224. Washington-New York: Robert B. Luce, Inc., 1971.

In her book, Dr. Whipple has supplied the medical profession, parents, and youth with much-needed information on the facts of drug use in our culture. She has asked pertinent questions: What does LSD do to the Ego? Does cocaine have any long time effects? What does psychedelic mean? What do barbiturates do to sleep? What is REM sleep? Is alcohol a psychoactive drug? If alcohol is legal, why not marihuana? What happens to those who get caught with marihuana? Are illegal drugs adulterated? What is the methadone maintenance treatment? How successful are these therapeutic community houses? What are the reasons given for the present drug laws? What does more harm—drugs or the laws against them?

Her answers are relevant. They are helpful to physicians, especially those involved with youth. Adults in general, parents specifically, need to be informed in order to bridge the communication gap between the generations. Factual knowledge on drugs leads to greater understanding of the youth culture and may alter attitudes of both adults and youth. And factual knowledge on drugs should be available to youth before a social experiment may lead to a serious tragedy.

Dr. Whipple traces the historical background of our commonly used drugs, their social and medicinal use in various cultures, their natural habitat, and their synthetic development in our clinical laboratories.

The complexity of our drug problem, however, develops through the variety of drugs often used in combination unknown to the consumer. Effects are unpredictable due to uncensored dose and dangerous combinations.

Physical and mental illness with often irrevocable addiction may ensue. The author has provided us with details. She shows that drug effects vary with the different personality as well as with the changing mood of the individual. Unstable and immature drug users have an unpredictable and often tragic outcome. A stable adolescent or mature young person in his twenties using marihuana, for example, may experience without harm a similar pleasurable sensation as his parents from alcohol.

The author gives a detailed inventory of all commonly used drugs. But independent of the pleasure that may be gained or the anxiety or tragedy felt by the mood or mind change, drug use is governed by law.

At present there is no comfort in being convicted in any of our states as can easily be seen in the quoted laws. Have any laws prevented drug use? Dr. Whipple says no. She draws on the experience of other countries, England for example, to prove that there are other ways than punishment to control drug addiction. She discusses methadone treatment. But considers as unrealistic attempts to pay foreign governments to stop production at the source.

We need a new look at our problem and a willingness to make necessary changes. Dr. Whipple has presented us with the facts—it is up to the concerned public to effect action—which in turn may develop a closer relationship and more understanding between the generations.

"Is The Grass Greener?" is an excellent book—a must for anybody who wants to be informed on the use and misuse of drugs.

CATHARINE KNOP, M.D.

Management of the Patient with Subnormal Vision, 2nd edition. GERALD FONDA, M.D. Price, $13.00. Pp. 167 with 114 illustrations. St. Louis: C. V. Mosby Company, 1970.

Anyone concerned with this problem will recognize this monograph as the product of enormous personal experience, and will appreciate the short cuts it provides. This book is physician oriented, emphasizing what the physician needs to know and do to assist the patient with subnormal vision: for example, how to classify patients and how to determine the most effective combination of illumination, visual aids and techniques for each patient.

The material is very well organized into fourteen chapters richly illustrated with well-chosen tables, diagrams and photographs.

ROBERT W. NEIDLINGER, M.D.

BOOK REVIEWS

Ultrastructure of the Peripheral Nervous System and Sense Organs, Atlas of Normal and Pathologic Anatomy. JEAN BABEL, ALBERT BISCHOFF AND HEINRICH SPOENDLIN. Price, $45.25. Pp. 452 with 203 illustrations. St. Louis: C. V. Mosby Co., 1971.

The truth of the Chinese Proverb, "One picture is worth ten thousand words", applies to few branches of medicine as much as it does to the neurosciences. There are several good atlases illustrating the histoanatomy and histopathology of the nervous system as illustrated by light-microscopy, and the classic German language text of Hager (1964) contains a number of small electron microscope photographs. However, continued advances in the techniques of electron microscopy and increases in knowledge generated by its application have created the need for a good general atlas illustrating the fine structure of the nervous system and sense organs.

The present work succeeds admirably in filling this void. The authors are a Swiss neurologist, otologist, and ophthalmologist respectively, each of whom has been responsible for a separate section dealing with his own specialty. The section on the peripheral nervous system is divided into illustrations of normal anatomy, general cytopathology and histopathology, and a collection of electron micrographs of the changes found in peripheral nerves damaged by various metabolic, toxic, and hereditary diseases. The sections on otology and on the retina follow a similar division into normal anatomy and pathologic changes.

The book is well arranged, with text and line-drawings or photomicrographs on the left page and the electronmicrograph of the equivalent structure on the right page. The text is succinct, clearly translated, and relates well to the pictures. The quality of the electronmicrographic illustrations is superb, and they are provided with numerous well-placed markers and abbreviations. The meaning of most of these is clear from a glance at the accompanying text, but a full glossary is also provided. Many pictures occupy an entire page, and their clarity and distinctness of detail speaks highly of the skill both of the authors' are printers' technique. As lagniappe for the reader, many of them are quite beautiful.

Though expensive, the work is reasonably priced for its quality. The sampling of basic structures will interest workers in many different fields and should appeal particularly to medical students in their basic science years, and to residents in neurology, neurosurgery, otology, and ophthalmology. This book should find a niche in every medical library.

RAMON JENKINS

Symposium on Horizontal Ocular Deviations. DONELSON R. MANLEY, M.D. Price, $26.50. Pp. 235 with illustrations. St. Louis: C. V. Mosby Company, 1971.

There has been a proliferation of published symposia in recent years, often of no lasting value. Fortunately, in this volume Dr. Donelson Manley, through painstaking efforts, has created a first class text book covering horizontal strabismus. The members of the Symposium have re-written their original papers to provide a thorough and well organized reference book. The book begins with a lengthy, very basic and very detailed section on esodeviations. This covers classification, diagnosis and treatment and would be of great value to the Resident in Ophthalmology. Sensory adaptions and sensory anomalies in strabismus are then handled with considerable clarity. The surgical problems of esotropia are then discussed. Attention is given to surgical failures, secondary procedures and newer techniques such as plastic implants to reduce adhesions. Exodeviations are given almost equal treatment. It would be difficult to find any single reference to equal this in its discussion of exotropia. The book is slightly uneven, possessing chapters that are very elementary and chapters that are esoteric but this does not detract much from its basic quality.

Horizontal strabismus is the overwhelming ocular motility problem facing Ophthalmologists and the Pediatrician. I know of no other book that has grouped such illustrious specialists to create a complete summary of available knowledge in this field. This is a good basic text, a good review and will be a good guide to the practitioner.

DAVID K. BERLER, M.D.

medical annals of the district of columbia

vol. 41, no. 1 january, 1972

Teeth, Teeth, Teeth: A Treatise on Teeth and Related Parts of Man, Land & Water Animals from Earth's Beginning to the Future of Time. SYDNEY GARFIELD, D.D.S. Price $9.95. Pp. 448 with illustrations. New York: Simon and Schuster, 1971.

Dr. Garfield merits a rousing "A" both for effort and for ambition in his herculean undertaking to expatiate on teeth "from Earth's Beginning to the Future of Time." Obviously, such a vast existential continuum cannot possibly be compressed within a span of 448 pages; therefore, his hyperbolic claim, however well-intentioned, must be relegated to the category of "Mission Impossible."

Despite this caveat, the author is to be admired for the expansive spectrum of research that rendered possible the volume under review. His diligence is reflected in the considerable corpus of valuable information that rewards the reader. However, the proliferation of subjects treated in Dr. Garfield's 23 chapters, at times militates against their receiving ample discussion.

To illustrate: Chapter 1 presents an illuminating 40-page exposition on "The History of Dentistry;" Chapter 3 on Psychosomatic, Psychological, Psychiatric Dentistry—Mental Factors, Fear of Pain and Sex Related Symbolism," is given short shrift in a sparse nine pages. This thumbnail sketch can hardly be called adequate.

On the positive side, the Table of Contents includes a veritable treasure-trove of significant topics in dentistry: Examination; Diagnosis and Treatment Planning; Anesthesia; Peridontia; Endodontia; Oral Surgery; Pedodontia; Orthodontia; Fixed Prosthodontics—Jackets, Bridges, Splints, Implants; Partial Removable Dentures; Full Removable Dentures; Nutrition.

Hundreds of Photographs and drawings, together with more than 80 pages of art illustrations, (which include three pieces of sculpture by Dr. Garfield), provide graphic visual commentaries to the textual material. A bibliography of dental literature, together with a helpful 6-page index, serve to round out this encyclopedic treatise, intensely researched and lucidly written.

The last illustration on page 441, which depicts Don Quixote in a colloquy with Sancho, is an appropriate envoy to the over-all material in Dr. Garfield's volume. It includes these lines from Cervantes' novel; "For I had rather have lost an arm provided it were not my sword arm. For I would have you know, Sancho, that a mouth without molars is like a mill without a stone and a tooth is more precious than a diamond."

SIDNEY N. GREENSPAN, D.D.S.

The Clinical Recognition of Congenital Heart Disease. JOSEPH K. PERLOFF, M.D. Price, $25.00. Pp. 606 with 578 illustrations. Philadelphia: Saunders, 1970.

This excellent textbook is exceptional in that the format is well organized, is precise, unusually well illustrated with meaningful material and each of its 31 chapters is followed by a listing of well selected current references. Each chapter is devoted to a specific anatomical or functional defect beginning with simple congenital positional anomalies of the heart and great vessels. Appropriate space is given to the common defects with a minimal but adequate discussion given to the clinical features of the rare and uncommon defects.

For once, an author selects a title and then prepared a text to fulfill the requirements of the title. This is truly a text devoted to the clinical recognition of congenital heart disease, not only that seen in infancy but relates to the clinical features and diagnosis during the natural course of the disease and life span of the individual, including the usual complications seen in infancy, childhood and adult life. No mention is made of embryology, therapy, prognosis, etc.

The author has an excellent background to prepare such a text, having broad training and experience in both pediatric and adult cardiology. Being an astute clinician and gifted with the ability to precisely translate what he observes, sees and hears to others, this textbook, the product of his clinical experience becomes a useful tool and not just a reference book.

Outstanding features of this text are: (1) the development of a classification of defects based upon the anatomical fault and physiological disarrangements rather than the usual classification by syndromes and name diseases, thus permitting a basic understanding of the anomaly and (2) the "synthesis" or correlation of the clinical and laboratory features leading to a logical diagnosis. Minimal reference is made to such syndromes and only that sufficient to orient the individuals bred on syndromes. This latter feature and simplification above is worth many times the price of the text, especially to the student and younger physician who has not yet become confused in this rather simple area of cardiology by the use of meaningless name disease and syndromes.

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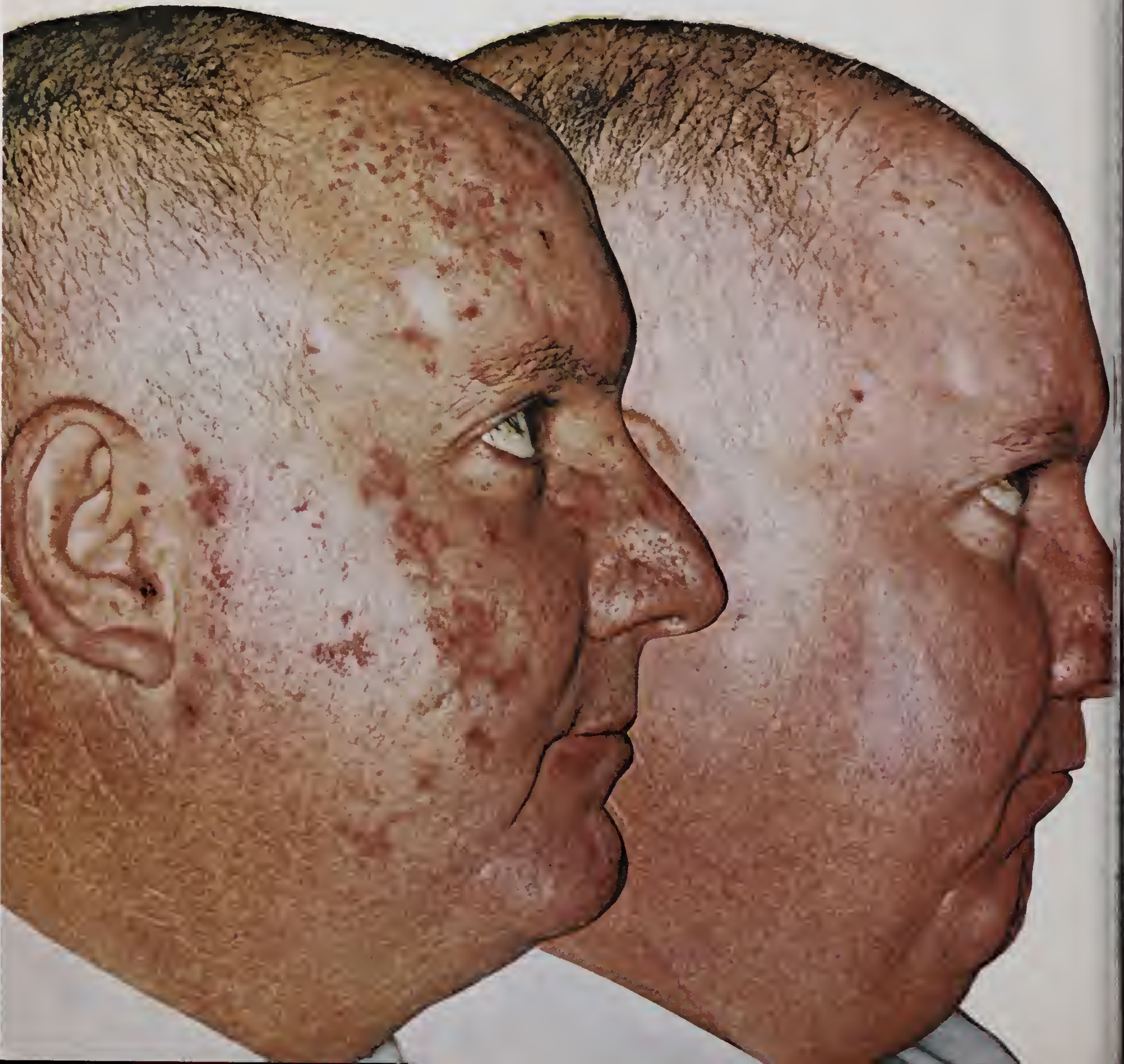
3/29/67 Before therapy with 5%-FU cream. Patient P. T. shows a moderately severe solar keratotic involvement. Note residual scarring from the previous cryosurgical and electrosurgical procedures on forehead and ridge of nose adjacent to periauricular area.

After

6/12/67 Seven weeks after cessation of therapy. Reactions have subsided. Residual scarring is not seen except for that due to prior surgery. Inflammation has disappeared and face is clear of keratotic lesions.



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ifested themselves by definite reactions, while intervening skin
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response to a typical course of Efudex therapy is usually
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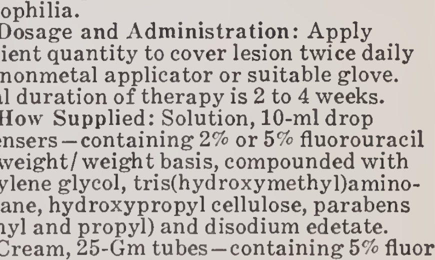

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*Data compiled from reports of 14 investigators. **Diagnosis was confirmed by cultural identification of *N. gonorrhoeae* on Thaye Martin media in all patients. Criteria for cure: negative culture after at least 2 days post-treatment in males and at least 7 days po treatment in females. Any positive culture obtained post-treatment was considered evidence of treatment failure even though t follow-up period might have been less than the periods cited above under "criteria for cure" except when the investigator determin that reinfection through additional sexual contacts was likely. Such cases were judged to be reinfections rather than relapses failures. These cases were regarded as non-evaluatable and were not included.

JA72 184

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globin, hematocrit and creatinine clearance; elevation of alkaline phosphatase, BUN and SGPT. In single and multiple-dose studies in normal volunteers, a reduction in urine output was noted. Extensive renal function studies demonstrated no consistent changes indicative of renal toxicity.

Dosage and administration: Keep at 25°C and use within 24 hours after reconstitution with diluent.
Male—single 2 gram dose (5 ml) intramuscularly. Patients with gonorrheal proctitis and patients being re-treated after failure of previous antibiotic therapy should receive 4 grams (10 ml). In geographic areas where antibiotic resistance is known to be prevalent, initial treatment with 4 grams (10 ml) intramuscularly is preferred.
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nd hypersensitive to Trobicin. Not indicated for the treatment
yphilis.

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Warnings: Antibiotics used to treat gonorrhea may mask or delay the symptoms of incubating syphilis. Patients should be carefully examined and monthly serological follow-up for at least 3 months should be instituted if the diagnosis of syphilis is suspected.

Safety for use in infants, children and pregnant women has not been established.

Precautions: The usual precautions should be observed with atopic individuals. Clinical effectiveness should be monitored to detect evidence of development of resistance of *N. gonorrhoeae*.

Adverse reactions: The following reactions were observed during the single-dose clinical trials: soreness at the injection site, urticaria, dizziness, nausea, chills, fever and insomnia.

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Adverse Reactions: Principally gastrointestinal; unpleasant metallic taste, continuing to anorexia, nausea and, less frequently, vomiting and diarrhea. Reduce dosage at first sign of these symptoms. In case of vomiting, the drug should be immediately withdrawn. Although rare, urticaria has been reported, as have gastrointestinal symptoms such as anorexia, nausea and vomiting following excessive alcohol intake.
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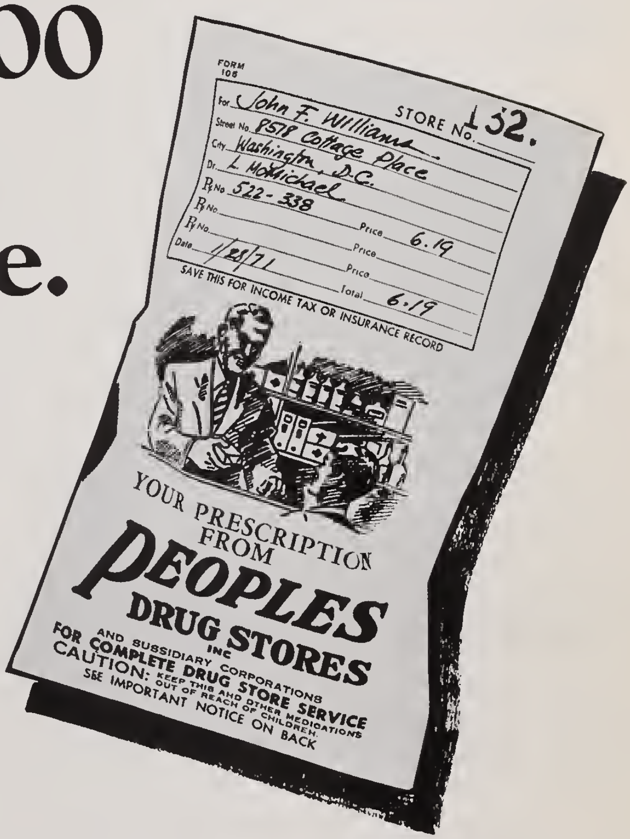
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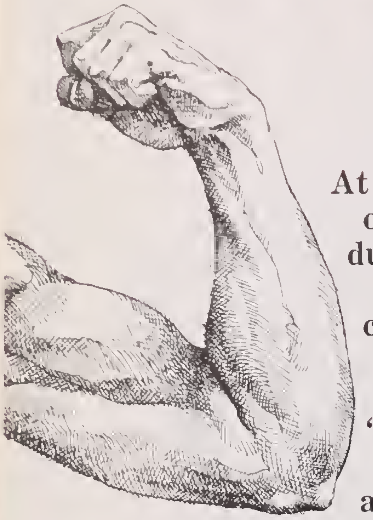
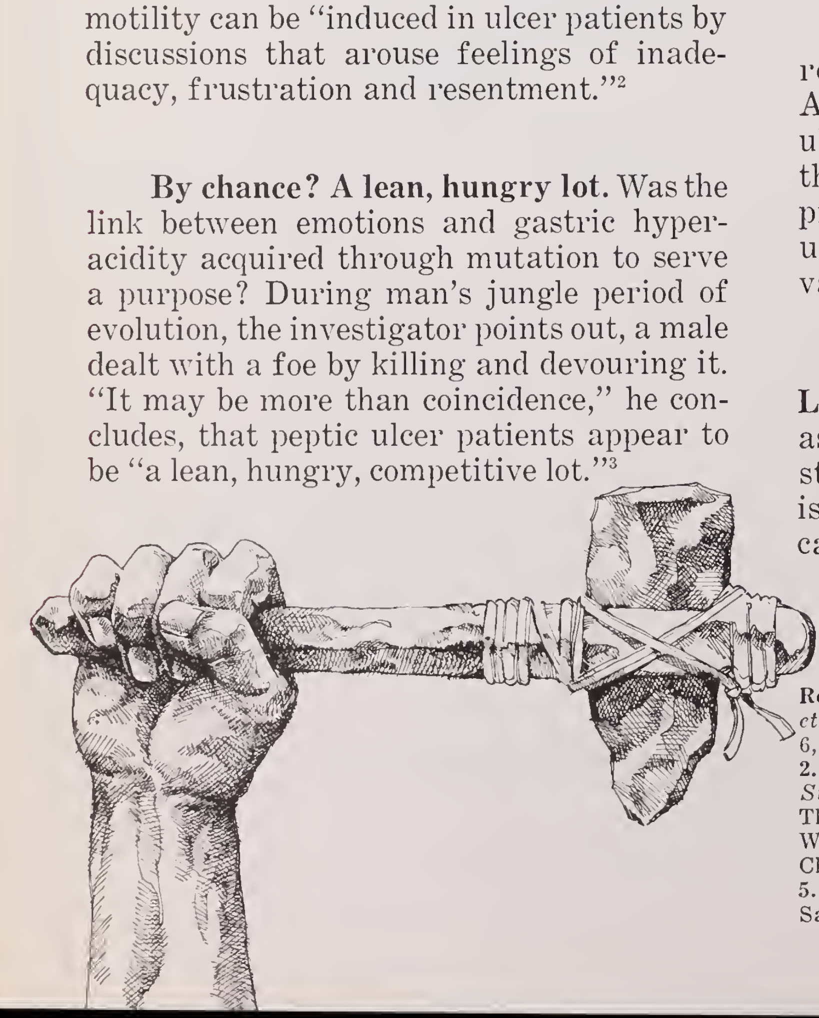


At least seventy-five out of one hundred adults with duodenal ulcers are men.[1] Why? It may be significant that duodenal ulcer patients tend to crave recognition and are "especially vulnerable to threats to their manly assertive independence."[2]

Hypersecretion—an atavistic response. Stewart Wolf, who, with Harold G. Wolff, studied the personalities of duodenal ulcer patients, wonders if masculine competitiveness is related to "an atavistic urge to devour an adversary." It is striking, he reports, that an accentuation of gastric acid secretion and motility can be "induced in ulcer patients by discussions that arouse feelings of inadequacy, frustration and resentment."[2]

By chance? A lean, hungry lot. Was the link between emotions and gastric hyperacidity acquired through mutation to serve a purpose? During man's jungle period of evolution, the investigator points out, a male dealt with a foe by killing and devouring it. "It may be more than coincidence," he concludes, that peptic ulcer patients appear to be "a lean, hungry, competitive lot."[3]



Big boys don't cry. If more men crie
maybe fewer would wind up with duodena
ulcers. But men will be men—the sum total
their genes and what the
are taught. Schottstae
observes that when
mother admonishes h
son who has hurt himse
that big boys don't cry, sh
is teaching hi
stoicism.[4] Crying is th
negation of everythin
society thinks of as manl
A boy starts defending h
manhood at an early ag

Take away stres
you can take away symptom

There is no question that stress plays
role in the etiology of duodenal ulce
Alvarez[5] observes that many a man with a
ulcer loses his symptoms the day he shuts u
the office and starts out on a vacation. Th
problem is, the type of man likely to have a
ulcer is the type least likely to take lon
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et al. (eds.): *Harrison's Principles of Internal Medicine,*
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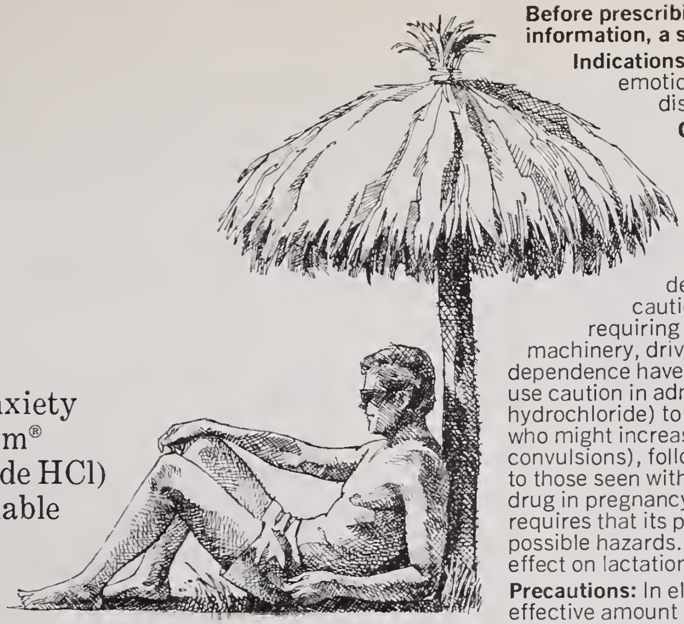

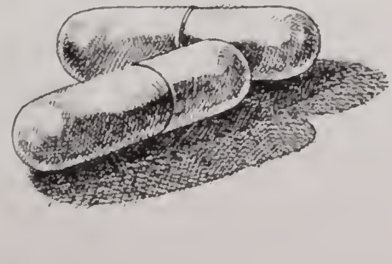

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Indications: Indicated as adjunctive therapy to control emotional and somatic factors in gastrointestinal disorders.

Contraindications: Patients with glaucoma; prostatic hypertrophy and benign bladder neck obstruction; known hypersensitivity to chlordiazepoxide hydrochloride and/or clidinium bromide.

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Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude development of ataxia, oversedation or confusion (not more than two capsules per day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

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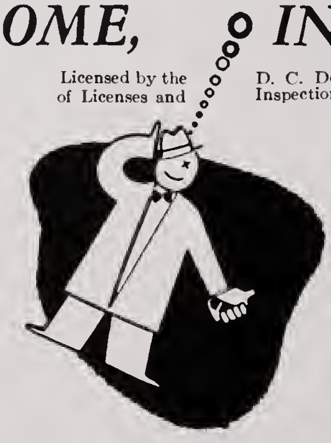

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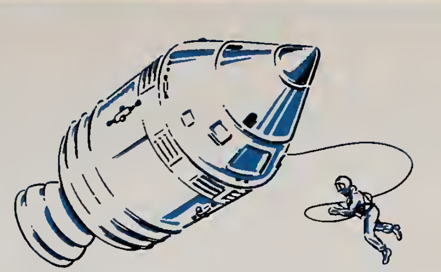

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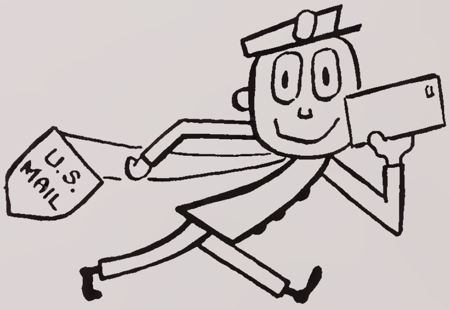

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The most commonly reported side effects are drowsiness, ataxia, and fatigue. Until individual response is determined, caution patient against driving or operating dangerous machinery.

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Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence. In pregnancy, lactation or women of childbearing age, weigh potential benefit against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodi blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiet and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed ar tolerated (not for use under 6 months).

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PRESIDENT'S MESSAGE

NEWS: *"Something strange or newly happened."*
"Matter adopted to gaining the interest of newspaper readers."

Webster's Unabridged Dictionary

"The news is what I say it is."

David Brinkley

"I fear three newspapers more than 100,000 bayonets."

Napoleon Bonaparte

"Every newspaper editor owes tidbits to the devil."

La Fontaine

Darrell C. Crain, M.D.

If misery loves company, the many physicians who were angered and incensed by the Washington Post headline story of January 4th dealing with physician refunds to Blue Cross may derive a modicum of solace from the realization that others, now and before times, have also suffered from the malevolent pens of the fourth estate.

Imagine the feelings of the President of the United States the very next day when he saw the private and personal conversation of the Internal Security Council blazoned across the paper by a columnist even his peers call "a muckraker".

Physicians have not - as some seem to feel - been singled out for individual vilification. Unfortunately, we are passing through a period in history when the iconoclast seems more popular - certainly more newsworthy - than the disciple. And because of the position of respect our profession has always enjoyed, we are prime targets for such attacks.

In the week following the newspaper articles many physicians spoke to me about them. All were indignant and two questions were repeatedly raised, "Why did the President of Blue Shield do this?" and "What is the Medical Society

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doing about it?". Mr. Victor Brian has presented his answer to the first question in a letter to participating physicians. It would appear that at a crucial moment in the ball game he rather naively took too big a lead off first base and was picked off by the quick pitch of a sharp cub reporter. The loser turned out to be not so much Mr. Brian as his supporting team.

Answers to the second question are of more significance. Your Society has notified Blue Shield that in the future, cases in which there appear to be irregularities in billing are to be submitted promptly to the Society for adjudication. If irregularities are determined, miscreants will be dealt with by the appropriate committees.

A letter was sent to the Editor of the Washington Post attempting to place the matter in proper perspective. The exact value of such communications is, of course, questionable, but they at least put the Society on record regarding facts - and reassure our friends among the newspaper reading public. We are planning to meet more frequently with members of the press to show them the diligent and conscientious work being carried on by Washington physicians.

Most important of all - new and important projects are being developed by officers and committee chairmen which when presented in the coming months will demonstrate to press and public alike the leadership which our Society has assumed in the matters of health care.

As President of our Society I am acutely aware of our members' dedication. I intend to make others aware of it too.

Required reading: *The News Twisters*
by Edith Efram
The Nash Publishing Company
Los Angeles, California

DOCTOR, PLEASE SEE MY PATIENT...

As the practice of medicine becomes increasingly complex, more and more patients are seen in consultation by one or more physicians. Unless handled properly, each of these requests provides an occasion for misunderstanding. The misunderstanding may be between the physicians or between the physicians and the patient.

Why does this happen? Why does a request which is intended to improve patient care deteriorate in this fashion? Lack of adequate communication is the answer here as it is in so many other areas of interpersonal difficulties.

In the past consultations were occasions when physicians truly consulted with each other about a patient problem. They met at the bedside and discussed the problem at hand. During this dialogue, the role of each physician in the patient's continuing care could easily be established. The complexity of private practice in a metropolitan area precludes that approach now. Since the personal contact is now almost always absent, the participants have obligations to each other which should be made clear.

The referring physician should indicate whether he is asking for a diagnostic opinion only or whether he also wishes a plan of management to be undertaken. In some cases the referring physician will wish the consultant to assume total responsibility for the patient. He should make that perfectly clear at the outset. The patient should always be told that another doctor has been asked to render an opinion. As elementary as that seems, it's amazing how often it's overlooked.

The consultant, for his part, has responsibilities too. Perhaps the most important is promptness. A hospital patient should be seen the same day if any urgency is involved and by the next day if the situation is not urgent. The consultant should leave his opinions and recommendations in a note on the patient's chart at the time of the consultation. Often a phone call to the referring physician is helpful since he may then request that the consultant write any orders which should be handled promptly.

Ordinarily the consultant should not discuss his findings in detail with the patient. He should rather indicate that his evaluation is not yet complete and that laboratory studies and X-rays must be reviewed before rendering a final opinion. In this way it can be made clear that the referring physician remains in charge of the patient's care and final responsibility for treatment rests with him.

Attention to these simple matters should result in better patient care and a decrease in interpersonal misunderstandings between physicians and patients.

Robert W. Langevin, M.D.

A NEW LOOK AT GENERAL HOSPITAL PSYCHIATRY

Prior to World War II the number of psychiatric units in general hospitals and the number of patients treated were relatively small. After the war, and probably because of the military experience of many psychiatrists returning to civilian practice, America experienced a great upsurge of interest in general hospital psychiatry. Psychiatric units doubled and tripled in number. Many of them became highly effective instruments for short term treatment. They rapidly became popular and were welcomed by the medical world and the lay public. It was predicted that there would soon be psychiatric units in most of the 5400 general hospitals in the country. These predictions have failed to materialize in spite of the demonstrated effectiveness of the psychiatric unit. By 1963 there were slightly less than 500 such units and an additional 500 hospitals admitted psychiatric patients to their medical wards without special facilities. Nevertheless, these general hospitals treated more psychiatric patients (370,442) during 1963, than did all the state and county hospitals combined!

The advantages of treating a patient in a psychiatric unit of a general hospital are many. It provides opportunity for immediate treatment in a familiar setting close to the patient's home and work. The stigma of the long term mental hospital is absent. Most patients enter on a voluntary or informal basis. The closeness to medical and surgical services is an important asset. Treatment of a wide variety of disorders is possible using the medical model in its best sense. Continuity of care following discharge is easily arranged. The psychiatric unit is geared to rapid treatment and rapid return to the community.

Then why hasn't American psychiatry taken even greater advantage of this modality? Several answers come to mind. Even though hospital governing boards and the lay community want to see more of these units, young men to spearhead them are not forthcoming. Present day psychiatric residency training programs tend to influence graduates away from hospital work and more toward exclusive preoccupation with one-to-one office therapy. Hospital work is more demanding, more hectic and less conducive to the well-ordered life. Cost of treatment in a psychiatric unit may be prohibitive to patients not covered by good insurance. Finally, some units overemphasize one type of treatment (e.g. EST or Chemotherapy) to the exclusion of other modalities.

What is the future of general hospital psychiatry? Recent surveys indicate a 35 to 40 per cent increase in the number of psychiatric units in general hospitals since 1963. Changing the emphasis during psychiatric training will do much to attract more young men with broader backgrounds into this important field where they can fill a very real need. The psychiatric unit has great potential as a training ground for general psychiatrists. The psychiatric unit of the future will develop closer ties to the community and to training centers. Although the psychiatric unit of the general hospital will never replace the long term mental hospital (public or private) it shows promise of becoming the treatment locus of first choice and of becoming a uniquely American contribution to the treatment methods of psychiatry.

Zigmond M. Lebensohn, M.D.

TOWARD A NATIONAL POLICY ON AGING:
TOMORROW IS ANOTHER DAY

The purpose of the Second White House Conference on Aging (November 28-December 1, 1971) was to build a national policy for the elderly of America. It turned out to be a bland, well-organized program that gave some psychotherapeutic benefits to the delegates but offered nothing substantial and immediate for old people. The Conference could only be viewed as a success by those who measure progress by snail's pace incremental improvements.

Health is one of the triad of factors crucial to old people along with income and housing. There was no clear demand for nor policy laid out to eliminate poverty and supply adequate housing for older Americans. There was a call for significant expansion in Medicare coverage --in the year when this Administration was cutting back benefits. There was broad support for a national health insurance plan with emphasis on chronic disease and nursing home care, so important in old age. But there was muted criticism of all the present bills under Congressional consideration that offer inadequate coverage in these respects. This is true of the union-backed Democratic Party bill of Senator Edward Kennedy and the Republican Administration bill of President Richard Nixon.

Conference delegates voted for a Commission on Mental Illness of the Elderly (now a bill in the Senate) to rectify the grave omission of the elderly from the original Commission on Mental Health that gave rise to the community mental health center program. Emotional and mental illnesses rise progressively with age. Organic brain disease and depression skyrocket in late life. Suicide attains its zenith in white men in their 80's. Fifty per cent of all suicides occur after 60.

Many many serious health concerns were not discussed --the quota system against the admission of Medicare and Medicaid patients in voluntary hospitals, the problem of arteriosclerosis and our national dietary habits, the absence of geriatrics as a major part of the medical school curriculum, our nation's often disgraceful nursing homes, and so forth.

President Nixon did announce an eight-point nursing home improvement program last summer. Health, Education and Welfare Secretary Elliot Richardson chose the midpoint in the Conference to threaten the withdrawal of funds from 37 states and the District of Columbia unless deficiencies were remedied by February 1. The test is here. Will there be grace periods or enforcement of laws that have been on the books since 1965 and not implemented by either the Democratic or Republican Administrations.

Final appraisal of the Conference must be done by examining the allocations of new monies in the federal fiscal 1973 budget. Can our nation tolerate any longer the poverty of our old people? Ten million of our 20 million elderly exist on $75 or less per week. Eighty-five per cent of the poor elderly are white and many have grown poor after growing old. The black elderly poor have one-half less income than the white poor. Six million of the 20 million live in substandard housing. Many are too poor to afford public housing. Medicare only covers 42 per cent of the health expenses of the elderly.

It is obvious that the creation of a national policy on aging demands a multi-billion commitment to our nation's elderly. It is unfortunately plain that this Conference did not and the Administration and the Congress will not take this giant step. Tomorrow is another day.

Robert N. Butler, M.D.
Chairman,
District of Columbia
Advisory Committee on Aging

SCIENTIFIC ARTICLES

A Quantitative Experimental Skin-Grafting Technique in Mice*

EDWARD J. BREYERE, Ph.D.†

The realm of immunology is constantly expanding to encompass many seemingly unrelated phenomena. The present state of knowledge could not have been achieved without the prior development of quantitative methods of measurement. Among various *in vivo* systems are those utilizing the survival of skin allografts (or homografts) as a measure of immunologic reactivity. For example, the abnormally prolonged survivals of grafts may be used to measure immunosuppressive effects of drugs for the transplantation of normal tissues, and the immunosuppressive effects of viral or chemical carcinogens.

Regarding the choice of a normal tissue for transplantation, skin offers several advantages: (1) it can be transplanted orthotopically, (2) observed directly for evidence of impending or complete rejection, and, if necessary, (3) biopsies can be readily obtained. A search of the literature will produce a variety of methods, but many will yield only qualitative information, i.e., either survival or rejection. Unfortunately, modern immunology demands entrance of the element of quantity. Varying degrees of alteration in host response require that survivals be quantified with a relative degree of accuracy.

Over the years we have developed a method of skin grafting that meets these requirements and offers certain advantages over that originally described by Billingham and Medawar.[1,2] Our modification has withstood the test of time and is being used in many other laboratories both in government and private industry. Two or more skin grafts may be placed in a single bed for quantitative determinations of their respective survivals,[3-5] and the method is also applicable to skin grafting in rats.[6] Since we have often been requested to furnish information concerning this method, we are presenting it here in detail.

The first consideration is selection of donor and host strains that will result in skin-graft survivals of greater than 10 days and which will allow survival to be determined by gross inspection. Highly incompatible genetic combinations resulting in shorter survivals necessitate histologic assessment of all grafts; in general, avoid incompatibilities at the H-2 locus that is similar to HL-A in man. The many inbred strains now available offer many possibilities, and 2 suggested combinations are CBA-C3H and BALB/c-DBA/2.

Preparation

Donors: Clip the hair from dorsum and sides of the body. Anesthetize with ether or other suitable anesthetic, and apply a depilatory (e.g., "Nair") to the clipped area. After a sufficient time, usually about 2 minutes, rinse animals under warm tap water, dry with a towel, and return them to their cages.

It is best to place no more than 4-5 such "prepped" mice in a single cage. Mice showing active hair growth must not be used. Ten grafts measuring 0.8 to 1 cm in diameter can be obtained from a relatively large area of this size. Individual donors will serve as donors for both test and control animals since apparent variation in antigenicity of skin between individual mice is sometimes encountered. It is best to "prep" 1 or 2 spare donors in the event hair growth occurs or the donors fight and damage one another's skin.

Recipients: With the mice under anesthesia, shave either the right or left thoracic wall of each recipient with an injector-type razor blade. Use hot water and a non-toxic soap, and not soaps containing such things as hexachlorophene because they are toxic to mice. Shave an area about the size of a postage stamp. Remember that since grafts will be held in place by pressure applied over the rib cage the area must extend anteriorly to the forelimb. Most beginners make 2 mistakes; (1) they shave the abdominal area, and (2) they scarify the skin with the razor blade. Hold the blade almost parallel to the body surface and shave a mouse as if it might shave itself, i.e., with short rapid strokes. Plenty of hot water, soap, and a sharp blade are essential. Return mice to cage, but no more than 5 per cage. In crowded cages mice are more likely to gnaw one another's bandages. Prepare both test and control recipients. We usually allow a day between shaving and grafting, and on the day of grafting we have the mice placed in clean cages. Using the skin from a single donor, plan to alternately graft-test and control animals so as to cancel out any individual variation in survival of donor skin. Be sure to ear-number or otherwise identify test and control animals prior to grafting. During the actual grafting procedure aseptic precautions must be observed.

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* This work was supported by Public Health Service Research Grant No. CA 05660 from the National Cancer Institute.

† Director, Immunogenetics Laboratory, Sibley Memorial Hospital, 5255 Loughboro Rd., N.W., Washington, D.C.; Professor of Biology, American University.

Grafting Procedure

Anesthesia: Dilute veterinary nembutal 1:9 with distilled water and store in stoppered vials under sterile conditions. Donors are given a large dose (about 0.4 ml depending upon the animal's size). If possible give nembutal i.v. slowly until a sufficient level is reached. This is the most direct way and precludes weighing the animals to calculate dosage. The recipients are injected (either i.p. or i.v.) with 0.15 ml of diluted material. During the operation this is supplemented with ether. After nembutal is injected the mice will not be fully anesthetized, but 1 exposure to ether in a jar will result in animals reaching a surgical stage of anesthesia that will often last for the entire operation. Supplemental ether is administered when necessary by ether-soaked cotton in a small vial which is placed over the nose and face. Under no circumstances should chloroform be used. Even in low concentrations it will produce fatal kidney damage.

Donor: Place a completely anesthetized animal on the operating board and fasten the legs in an outstretched position by means of clamps, tape, or pins. Sponge with alcohol, and by means of scissors and forceps make 2 lateral cuts, 1 across in front of the base of the tail, the other across the anterior area of the cleared ("Naired") skin. Make a third cut in an anterior-posterior direction. These 3 cuts allow a large rectangle of skin to be raised which is still attached at 1 side. The free end of the flap is then held until the end of the operation in a large clamp, e.g., Rochester-Pean. Now with the flap of skin raised, take a scalpel and clean away all blood, fat, muscle, and connective tissue from the skin (Fig. 1). Theoretically all you should have left is epidermis and dermis. Return the skin flap to the mouse. The animal's body makes the best medium for preserving skin while the operation is in progress. In this way the use of culture dishes and media can be avoided. Avoid drying by not exposing skin to the air for very long at a time.

In an effort to standardize graft size, a cork borer can be used to make an impression on the skin which is then cut with curved iris scissors. After the grafting technique has been perfected a few grafts can be cut beforehand and placed raw-side down in a sterile petri dish containing a few mls of saline. After all usable skin has been removed the donor is killed either by an overdose of anesthetic or cervical dislocation.

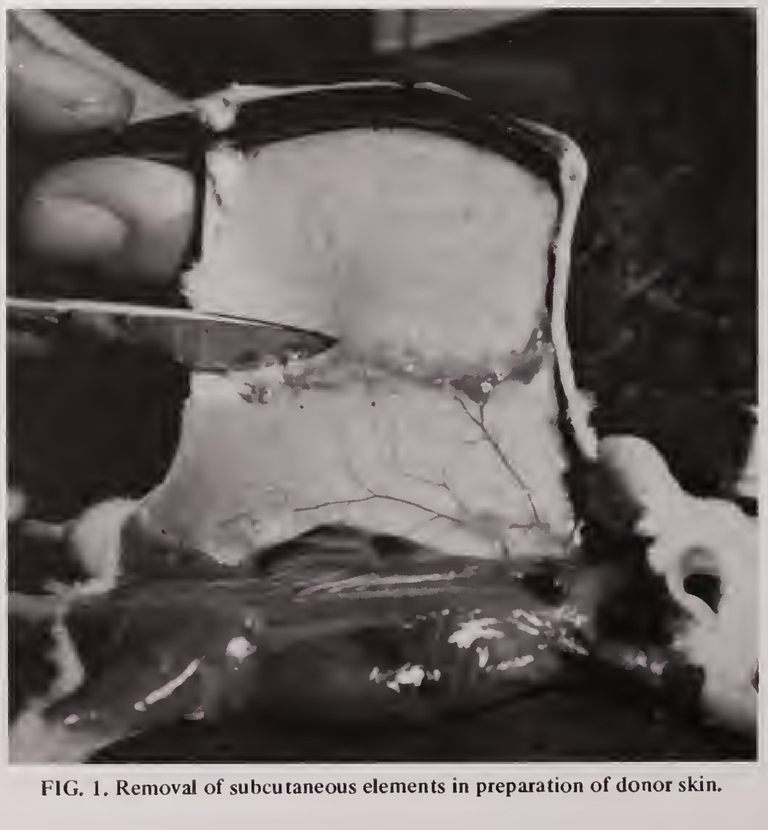

FIG. 1. Removal of subcutaneous elements in preparation of donor skin.

Recipient: Sponge shaved area of anesthetized animal with alcohol, and with a pair of very fine iris scissors prepare a graft bed by cutting through the epidermis and dermis down to the level of the connective tissue overlying the panniculus carnosus muscle. If bleeding occurs, hold the severed vessels a few moments with a small clamp (e.g., Halstead mosquito). Grafts should not be placed on clotted blood since they will not heal in properly. The procedure is done by snipping off small thin strips of skin. These are wiped from the scissors with a sterile gauze pad. Do not prepare beds by merely lifting up a tent of skin and cutting it free with scissors, since the graft bed will now be the rib musculature. Grafts made on such a bed are not in a normal anatomical relationship and the host skin will move while the graft remains stationary. Such grafts will often slip from their beds and result in technical failures. When a graft is placed upon a properly made bed it should move when the adjacent host skin moves. Prepare a graft bed much larger than necessary to accomodate the actual size of the graft—the larger the bed the fewer the technical failures. Place graft(s) in the bed, and since grafts will tend to fold in many places, straighten (raw side down!) with a pair of fine milled tip forceps (Fig. 2). Apply 2 layers of squares of vaseline-soaked gauze over the graft (about 1.2 cm^2). Press them down firmly with a pair of heavy milled-tip forceps. Close the forceps, place them in the center of the graft, and allow them to slowly open outwards. Note that this movement flattens the graft even more (Fig. 3). Cover these squares with a dry sterile gauze pad of similar size. Vaselined and plain gauze pads are prepared and sterilized beforehand in petri dishes.

Now for the most critical part of the graft procedure. Cut a ¼″ width strip of adhesive tape about 4½″ long. Place the center of the tape on the gauze pad and wrap the tape tightly

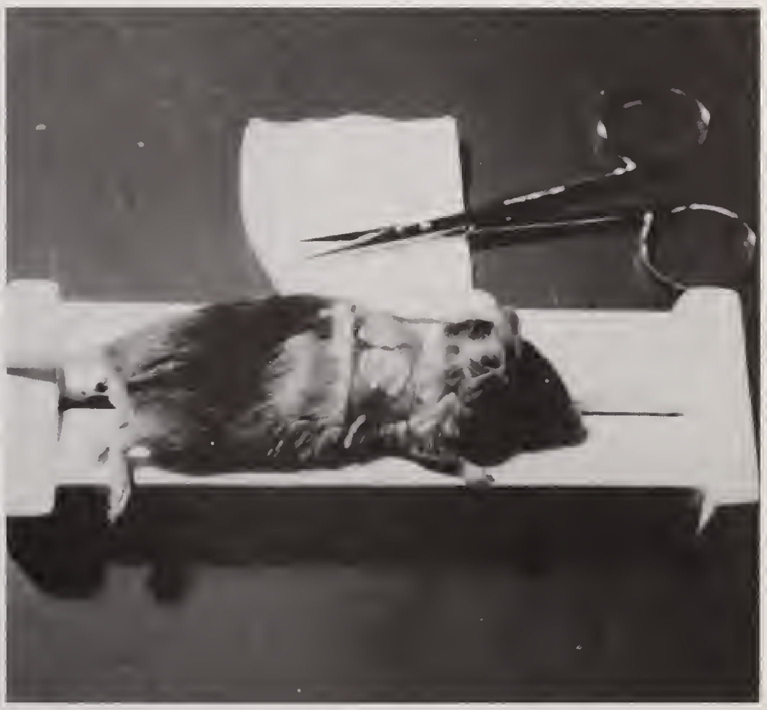

FIG. 2. Two grafts in place on bed prepared with fine iris scissors.

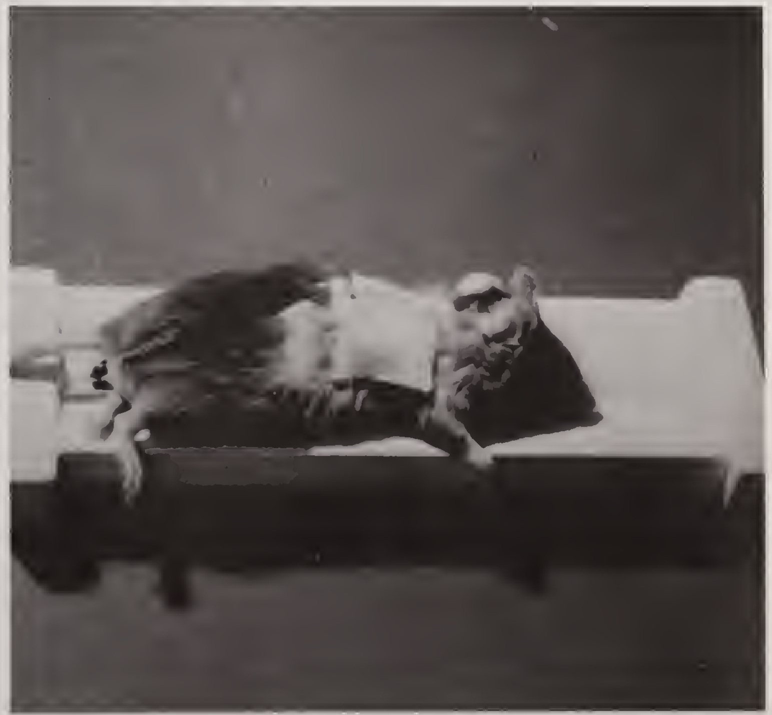

FIG. 3. Two layers of vaseline-soaked gauze (tulle gras) in place over grafts.

around the animal so as to exert pressure on the graft. Exert just enough pressure so that the animal's breathing is not impaired (Fig. 4). Most beginners do not wrap the animal tightly enough, so that the graft slips from the bed and is lost. If the bandage appears loose, stop and re-bandage. One good graft is worth the effort; questionable or technically bad grafts are worth nothing.

Cover these bandages with about 5 turns of ½" width roll gauze. Hold this in place with several turns of ½" width adhesive tape. Make sure that the tape extends over the gauze onto the hair of the host; the latter will help hold dressings in place. If these outer bandages appear too tight at the edges, cut small slits to relieve pressure. Also, if necessary cut away adjacent outer bandage to allow free movement of forelimbs. The outer bandages serve only to keep out dirt and protect inner dressings.

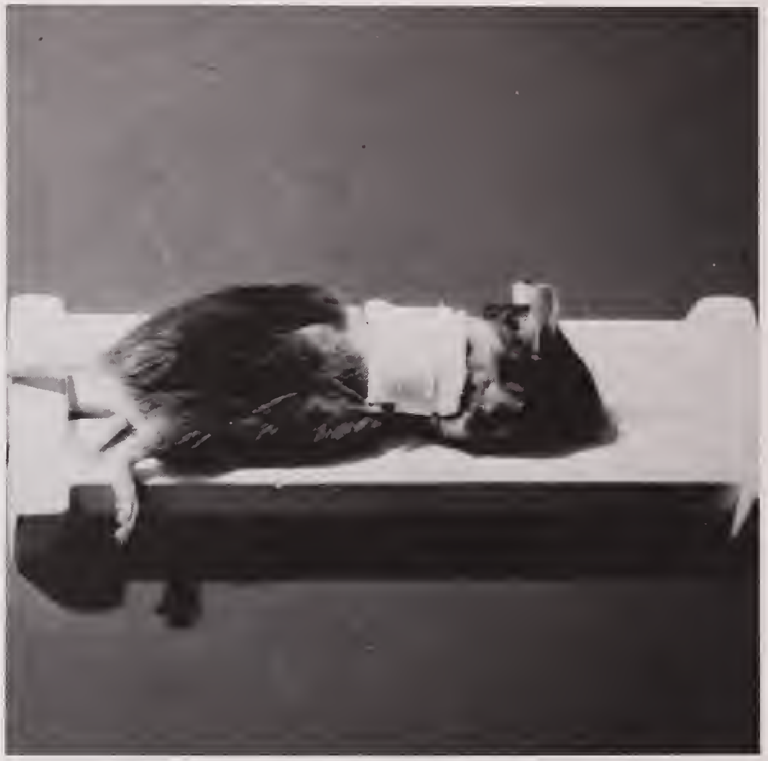

FIG. 4. Pressure applied to grafts by means of gauze pad and adhesive tape.

On the seventh day after grafting remove the outer bandage. Anesthesia is not required, but be careful not to hold the animal too tightly so as to impair breathing. On the ninth day remove the inner bandage and expose the grafts. Do this under ether anesthesia. Remove the gauze squares very carefully; grafts often adhere to them and can be torn loose when they are removed. Contracture of host skin around the graft often is not complete, and some bleeding may occur when the inner dressings are removed. If this happens, re-bandage, but only with a few turns of ½" width roll gauze covered with ½" width adhesive. Usually when these dressings are removed on the tenth postoperative day, no further bleeding will occur.

Scoring Graft Survival

Tables are constructed showing the animal number along one axis and the postoperative day and the date on the other. Grafts should be examined daily for the first month and every other day thereafter. In long-surviving grafts once a week is sufficient. This can be done (extending inspection intervals) since the pace of the rejection process is inversely proportional to the survival. Grafts are scored according to 2 characteristics: (1) the area of the graft still surviving (based upon its original area when the bandages were first removed on the ninth postoperative day), and (2) the appearance of the graft.

Area scores: + (50-100% survival), ± (less than 50% of the original area surviving), and – (0% survival).

Survival endpoint is taken as the last day upon which a score of ± was given or on the day the survival became questionable. Grafts undergoing rejection should be observed daily.

The appearance of grafts must also be recorded to serve as a guide on donor-host compatibility and strength of the rejection process.

Gross appearance: H (hair growth. Rarely observed before the third week after grafting. Indicates good compatibility, i.e., lack of immune response.), HW (white hair. Loss of pigment is often observed with genetically compatible grafts and may or may not be an indication of incompatibility.), and SW (swollen graft. Noted particularly in incompatible grafts due to edema in the dermis. This is often seen around the ninth postoperative day and prior to hair growth. This is often the first indication of an immune response.) PN (peripheral necrosis, scabbing at the junction of the graft and host skin. A sign of weak incompatibility.), and SN (spotty necrosis, scabbing over the graft surface. This is a sign of strong incompatibility.)

In most instances these necrotic areas will become larger and coalesce to include the entire graft surface, at which time the graft will be graded as "rejected" (non-viable). In rare instances when weak incompatibilities exist, these areas may heal over and rejection may begin again at a later time. This cyclic form of rejection has been reported but does not occur very often. These abbreviations minimize the length of information required in tables and make them easier to read. For example, the notation + HSW means that a surviving graft exhibits hair growth but is edematous, which probably heralds its impending rejection.

Analysis of data: Many authors have used probit analysis, and more recently the Mann-Whitney U test has been employed. However, Student's "t" test will give respectable estimates in terms of probability of differences between test and control groups.

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A Follow-up Study on Tuboplasties

SIMON SOLANO, M.D.*, RICHARD J. SCHROTT, M.D.† AND DOMINICK A. MINOTTI, M.D.‡

The European surgeons of the 1800's are credited with the fundamental techniques involved in reconstructive surgery of the oviduct.[1] Watkins[2] (1896) in the United States is said to have performed the first utero-tubal implantation. But gynecologists have traditionally looked at such surgery in the infertile woman with suspicion, especially after Greenhill's classic study in 1937 of 818 such operations, which yielded only a 6.6% pregnancy rate.[3] Siegler[4] has confirmed a reluctance to such surgery in a study which showed that only 15% of gynecologists have performed 10 or more tuboplasties.

Concerning the magnitude of the problem, it is said that as high as 1 in 10 marriages are sterile, and that tubal malfunction is the etiology in 1 in 6 of these cases.[5]

The present study was a follow-up done on 26 patients who had oviduct reconstructive surgery performed at Georgetown University Hospital between 1955 and 1969. In all of the patients the chief complaint was infertility. The classification of tuboplasties that is used here is the traditional one of salpingostomy, salpingolysis, utero-tubal implantation, end-to-end anastomosis, and combination procedures. A follow-up study of such operative procedures is presented here in terms of the ultimate objective, that is, a postoperative pregnancy, and this information has been correlated with the history and pathological reports.

The hospital charts of those patients who had tubal reconstructive surgery because of infertility were assembled. Pertinent features of their history, present illness, and follow-up were recorded on an information sheet. The follow-up questions were answered either by the patient herself or her gynecologist. Out of a total of 38 suitable cases a follow-up was obtainable on 26 patients.

Among 26 patients, 4 subsequently became pregnant following surgery, a 15.4% pregnancy rate. Two of the 4 patients had pregnancies which terminated in abortion, both women being multiparous and having had abortions. One patient delivered 2 term-pregnancies following a salpingostomy for bilateral tubal occlusion. The other live birth was the product of a nulliparous woman, who had a combination right salpingostomy and left partial resection and utero-reimplantation procedure. She had a history of chronic pelvic inflammatory disease and had a demonstrable left tubal occlusion (table 1).

The operative procedure most frequently preformed was a combination procedure, which was usually a bilateral tuboplasty, a different operation being executed on each oviduct. The most successful procedure was the salpingostomy, which usually involved opening and reconstructing the distal one-third of the tube by various techniques (-3 with Mulligan Hood applications) (table 2).

In the available pathological reports, only 1 patient had a tissue diagnosis of normal oviduct. The most prevalent condition was chronic salpingitis (table 3). The greatest number of patients had had acute or chronic PID (table 4). The majority of patients were nulliparous (table 5).

TABLE 1: Post-Operative Pregnancy Cases

Patient	Procedure	History	Findings	Results
1. J. G. g0p0	Combination right salpingostomy, left utero-tubal re-implantation	Chronic PID	Left tubal occlusion	1 live-birth
2. M. F. g4p1ab3	Salpingostomy	Chronic PID	Left hydrosalpinx	1 abortion
3. E. J. g3 p2 ab1	Salpingolysis	None	Left tubal occlusion	1 abortion
4. M. I. g1 p1	Salpingostomy	None	Bilat. tubal occlusion (clubbed fimbriae)	2 live-births

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Discussion

One of the most striking features of the data is the large number of patients with a history of pelvic inflammatory disease, and whose tissue pathology substantiated this history. Most students of this subject agree that a proper selection of surgical candidates is one of the most crucial steps toward a favorable result. Acute PID is said to be a contraindication to surgery,[1] and it is known that chronic PID lessens the chance of a surgical cure. The reason, it appears, is that a normal, physiologically functioning oviduct (especially cilia and fimbria) is necessary for "catching" and transporting the ovum to its final resting place in utero.[6] Artificial conduits for moving the ovum have been tried-a silicone ovisac,[1] a reverse ileal loop,[7] and an appendix,[8] but they have usually met with failure, presumably because they, like the scarred and inflamed oviduct seen in PID, do not possess those essential physiologic properties needed for movement of an ovum. And for these reasons it is said that the incidence of ectopic pregnancy is high following tuboplasty, although none appeared in this study.[9]

Because of the size of this series, it is difficult to make definitive judgments about the merits of each surgical procedure. But, in one of the best review articles on tubal surgery it was found that consistently best results were obtained by salpingolysis.[1] The apparent explanation for the relatively good results is that the procedure involves mainly blunt dissection of adhesions with little or no actual scar-producing tissue incisions, which one must do in and end-to-end anastomosis, for example. Shirkodar[10] thinks that one of the major reasons for failure of salpingoplastics is poor surgical technique. The plastic surgeon must consider the delicate anatomy of the oviduct.

TABLE 2: Operative Procedures

Procedure	No. Cases	Live Births	Abortions	Total
1. Salpingostomy	6	2*	1	3
(fimbrioplasty)				
(with Mulligan Hood's)	3			0
2. Utero-tubal implantation	3			0
3. End-to-end anastomosis	0			0
4. Salpingolysis	3		1	1
5. Combination procedure	11	1		1

* Both of these births were to the same patient.

TABLE 3: Pathology Report of Tubal Tissue

Diagnosis	No. Patients
1. Chronic salpingitis with or without hydrosalpinx	13
2. Salpingitis isthmica nodosa	1
3. Ectopic pregnancy	1
4. Paraovarian cyst	1
5. Normal tube	1
6. Unavailable reports	9

TABLE 4: Significant History

Condition	No. Patients
1. Acute or chronic PID	15
2. Uterine fibroids	4
3. Ovarian cysts	8
4. Previous tubal ligation	1
5. Diabetes	1
6. Sarcoidosis	1

TABLE 5: Obstetrical History

	No. Patients
1. Nulliparous	15
2. Term pregnancies (1 or more)	8
3. Abortions (1 or more)	6
4 Ectopic pregnancy (1 or more)	2

A proper pre-operative workup of the surgical candidate should be aimed at ruling out other causes of infertility and at demonstrating the tube as the etiology. Ideally, therefore, the husband should have a fertility workup, and the patient herself should have endocrine function tests when appropriate and show evidence of ovulation. To demonstrate tubal occlusion, culdoscopy or laparoscopy, Rubin's CO_2 insufflation test, or hysterosalpingography may be employed.

The incidence of pregnancy in this study was about 15%, which was similar to comparable studies, like that by Mroueh and Hajj[11] who reported a 10% pregnancy rate, and a Mayo Clinic study which reported a 24% pregnancy rate.[6] In general, the results of oviduct reconstructive surgery are not good, and even the very best results do not much exceed a 50% pregnancy rate.[1]

Summary

Twenty-six infertile women had oviduct reconstructive surgery. Four subsequently became pregnant, 2 with live births and 2 with aborted fetuses. The incidence of pregnancy was 15.4%.

Fifteen of the patients had a history of pelvic inflammatory disease. Pathology reports of tubal tissue confirmed this in 13 cases.

A careful selection of prospective salpingoplasty candidates and proper operative techniques are crucial to good post-operative results.

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Incus Interposition—A New Technique

JOSEPH A. SABRI, M. D.*

The incus is the most vulnerable ossicle in diseases of the middle ear and mastoid if otosclerosis is excluded. Its long process, being slender and poor in blood supply, is especially prone to caries and necrosis. Furthermore, the incus, being the intermediate ossicle with 2 articular surfaces, is prone to fixation when disease invades the epitympanum. With the advent of tympanoplasty several techniques were utilized to reconstruct the ossicular chain, incus interposition when feasible being most popular among otologists.[1-5] The current technique was to place the body of the incus over the capitulum of the stapes, supported with gelfoam, and anchor 1 of its processes under the manubrium of the malleus. This technique had many disadvantages because the incus was placed lateral to its normal anatomical position. The long process of the incus normally lies behind and mesial to the manubrium of the malleus. Placing the incus on top of the capitulum shifts its process laterally, thus creating difficulties in anchoring it under the manubrium. Quite frequently the tip eventually becomes adherent to the promontary. Also, there is no guarantee that the incus will stay flat over the stapes and in the healing process frequently becomes adherent to the adjoining structures. To overcome these disadvantages the following technique has been developed.

Technique

After the incus is removed from the tympanum the field is studied very carefully and prepared for interposition. The distance of the mobile stapes and malleus is measured carefully. If the malleus is fixed, its head is amputated, removed and saved. The stapedius tendon should be severed if the stapes does not project high enough above its attachment.

The incus, removed from the tympanum, is now grasped with the mosquito clamp, and under 10-power magnification a through-and-through perforation is made in its body with a Shea drill and a #4 burr. It is preferable to do the drilling under water, holding the incus in a cup filled with physiological saline. There is also an advantage in making the perforation funnel-shaped. The procedure is easy and should not take more than 5 minutes. After the perforation is made the incus is studied carefully, the shorter of its 2 processes removed, and a groove made at the extremity of the remaining process to fit under the manubrium of the malleus. The middle ear is inspected again, and if the field is dry the incus is then positioned so as to allow the newly created perforation to fit over the capitulum of the stapes giving it the appearance of a ball-and-socket joint (figures 1 and 2). This is achieved with the aid of 2 picks. Once the incus is secure and the capitulum of the stapes is seen to project through the perforation the process of the incus can be rotated like the hand of a clock with the aid of a pick and its grooved end positioned quite easily under the manubrium of the malleus.

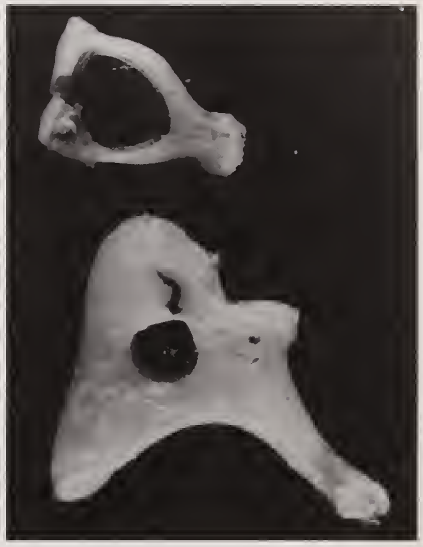

FIG. 1. Postmortem specimen illustrating the fenestrated incus.

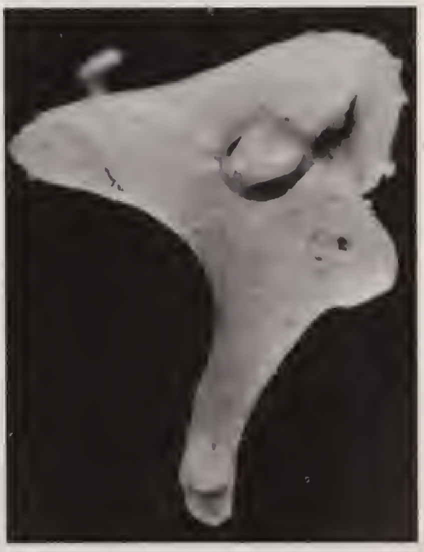

FIG. 2. The incus interposed over the capitulum of the stapes.

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The incus stays flat in this position and is completely secure, its process lying in the same plane as the manubrium of the malleous. No gelfoam is needed to support it. The chordotympani can now be placed on top of the body of the incus, and the surgeon can then proceed with the grafting.

Twelve patients were operated upon with this technique. Without exception all had closures of the air-bone gap to within 10 db. After 1 year all of them have shown no signs of deterioration in hearing.

Report of Three Illustrative Cases

Case 1. J. T., a woman aged 53, had a large perforation involving the posterior half of the tympanic membrane with dense tympanosclerotic plaques in the remaining part of the pars tensa and the pars flaccida. She had a hearing loss averaging 42 db by air with 12 db bone conduction. At surgery the incudomalleolar joint was found to be completely fixed with dense tympanosclerotic disease. The incus was therefore removed and a through-and-through perforation made in its body with a #4 burr using the Shea drill and was repositioned to fit over the capitulum of the stapes. The ear was grafted with temporalis fascia. Three months later the ear looked good with complete closure of the air-bone gap.

Case 2. A. A., a man 20 years of age, came with a history of bilateral otorrhea of 15 years' duration. He had almost total tympanic perforation, bilateral, with active granulation tissue filling the middle ear. After 2 months of medical treatment the left ear was operated upon. At this stage he had a 50 db average hearing loss in the left ear and 45 db in the right with bone conduction averaging 10 db. At surgery the lenticular process of the incus was found dehiscent and the stapes found fixed with dense connective tissue. The incus was therefore removed. A 1.5 mm hole was drilled through its body and a groove drilled close to the tip of its process to allow a good fit when positioned under the manubrium of the malleus. Fascia was used for graft. The patient had a complete air-bone closure.

Case 3. B. H., a 67-year-old woman, had a total tympanic perforation with a history of hearing loss of 30 years' duration. She had a dry ear with a hearing loss of 62 db average and 18 db bone. At surgery the long process of the incus was absent and the stapes was found fixed with dense adhesions. The incus was therefore removed and a perforation drilled in its body. The adhesions were removed from around the stapes, but these were so dense that while attempting to remove them there was a linear fracture in the footplate resulting in a slight perilymphatic leak which was sealed with gelfoam. The incus was then re-positioned in the usual manner and followed by grafting. The patient had closure of the air-bone gap up to 3000 frequency. However, she had a sharp drop between the 4000-8000 frequency range, presumably due to the perilymphatic leak and injury to the labyrinth.

The above cases are typical examples of this new technique. The remaining 9 patients on which a similar procedure was used had almost the same good results.

Summary

A new technique has been presented for incus interposition, the advantages of which are the creation of a ball-and-socket type joint between the capitulum of the stapes and the incus. This prevents dislocation or tilting of the incus. The fenestration in the incus allows its positioning more mesially, and its process falls in the same plane as the manubrium of the malleus and gives better anchorage.

There is no need to fill the middle ear with gelfoam to support the incus, thus diminishing the possibilities of future adhesions.

In case there is total destruction of the incus a homogram can be utilized in the same fashion. In my hands tympanostapidopexy has given poor results. When both the malleus and the incus are absent and the stapes is mobile a homogram fenestrated incus placed over the stapes and grafted will give better hearing result.

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Extracorporeal Circulation*†

ROBERT S. LITWAK, M.D.

The single most important advance in the surgical management of heart disease has been the development of practical methods of performing direct vision operations within the heart with temporary extracorporeal circulation. Clinical success with such a method was first accomplished in 1953 by Dr. John H. Gibbon, Jr., who had spent the preceding 16 years developing physiologic concepts and equipment to achieve a goal which all but a few thought impossible. With proper utilization of existing equipment a well executed operation and thoughtful post-operative care, an imposing list of cardiac abnormalities may be corrected in an unhurried fashion with considerable certainty that the patient will recover and be improved. Permissible periods of total cardiopulmonary bypass presently approximate 4 hours, although the vast majority of intracardiac operations are completed in less than half this time.

Equipment and Methods

Temporary cardipulmonary bypass is conducted with a variety of extracorporeal circuits all of which possess a means of pumping blood with minimal turbulence and a device for gas exchange of venous blood withdrawn from the patient so that oxygen and carbon dioxide tensions reflect those normally observed in pulmonary venous blood.

The most commonly used pump consists of 1 or 2 rotating rollers which compress a tube so that the contained blood is gently forced out. The output of these roller pumps is continuous and relatively non-pulsatile.

Three gas-exchange mechanisms ("oxygenators") are in current use. Dispersion of oxygen bubbles in blood with subsequent defoaming (bubble oxygenators) and surface filming of blood on either a series of stationary vertical sheets or rotating discs in an oxygen-rich environment are presently the 2 most commonly employed systems because of simplicity and the availability of cheap disposable units. The third type, a membrane "oxygenator" closely simulates conditions found in the natural lung by interposing thin gas-permeable membranes between the blood and an oxygen-rich atmosphere. This system minimizes protein denaturation which has been shown to be a consequence of the large blood-gas interface present when other "oxygenators" are used. Although early models were cumbersome and relatively inefficient, recent modifications have made these units increasingly practical for daily clinical use.

In practice, the heart-lung machine is usually primed with a mixture of heparinized blood and crystalloidal solution (hemodilution). Although conclusive data are lacking, clinical experience suggests that the diluted perfusate is advantageous because its lower viscosity improves tissue perfusion and diminishes blood trauma. The patient's blood is rendered incoagulable with heparin, and cannulae are inserted into the venae cavae or 'right heart' for diversion of venous blood into the extracorporeal apparatus. Return of oxygenated perfusate to the patient is accomplished with a cannula placed in the arterial circuit, frequently the ascending arch of aorta or a more peripheral vessel such as the iliac or femoral artery. At the onset of bypass, only a portion of the total venous return is diverted into the apparatus. This period of partial perfusion is used to check all portions of the circuit before definitive cardiac surgery is commenced. Total cardiopulmonary bypass is achieved by tightening snares around the caval cannulae, and the intracardiac surgery is begun. Intracardiac blood of coronary and bronchial circulatory origin is gently aspirated into the apparatus, defoamed, and returned to the patient. When the aortic valve is competent the perfusion pressure provided by the apparatus maintains the valve in a closed position and assures satisfactory bloodflow to vital organs. However, when the valve is distinctly incompetent or requires exposure for surgical correction, the ascending aortic arch is occluded, a proximal aortotomy made, and coronary artery cannulation and perfusion carried out. Blood (and therefore body) temperature is controlled by passing the perfusate through a heat exchanger. Moderate hypothermia (30–32°C) is often used to reduce total body-oxygen consumption although recently there has been an increasing tendency to conduct perfusions at normothermic conditions.

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Physiologic Requirements and Effects of Extracorporeal Circulation

The essential requirement of temporary extracorporeal circulation is that it satisfactorily replace the heart and lungs for a limited time. The need of the tissues for oxygen and the necessity for removal of carbon dioxide and other metabolites requires that the perfusion rate and gas exchange be sufficient for such purposes. Although basal cardiac output in normal subjects approximates 3.1 L/min/M^2, it has been found that flow rates of 2.2–2.4 L/min/M^2 are satisfactory for perfusion periods up to 4 hours. With these flow rates, maintenance of mean systemic arterial pressures of 50-70 mm Hg (in normotensive patients) is generally sufficient to assure satisfactory organ function during and after perfusion although reliable data concerning partition of bloodflow to organs during whole-body perfusion in man are not yet available.

An intra-operative fall in hematocrit, the consequence of perfusate dilution, is well tolerated despite the reduced oxygen-carrying capacity of the blood, provided that flow-rate, perfusion pressure, and gas exchange are properly maintained. Systemic arterial oxygen and carbon dioxide tensions must be kept at essentially normal levels (100 and 40 mm Hg respectively). It is particularly important that hypocarbia be avoided because of its adverse influence on cerebral function.

Stability of the intracellular and extracellular environments requires consideration of need for therapy of the reduced buffering capacity of the blood which accompanies significant hemodilution. Incremental infusion of buffers, either sodium bicarbonate or tromethamine, are generally used during perfusion. The latter offers the advantage of a non-sodium-containing ion which can traverse the cell membrane to provide both intracellular as well as extracellular buffering. Properly conducted, whole-body perfusion should not be associated with significant alterations in buffer base and hydrogen-ion concentration.

Cardiopulmonary bypass is presently associated with alterations in vascular volume, water and electrolyte distribution which may contribute to morbidity and mortality if not properly appreciated. There is a measurable reduction of blood volume, and the extracellular water is increased, although in the absence of pre-existing cardiac or renal failure this may largely be the result of intra-operative fluid administration during and after perfusion. Postoperative urinary excretion of sodium is low and potassium excretion high. Infusion of supplemental potassium (in the absence of oliguria) will normally prevent postperfusion hypokalemia and its attendant danger of cardiac arrhythmias.

Complications of Extracorporeal Circulation

Presently, the risk of cardiopulmonary bypass is remarkably low, perhaps 1% for perfusions lasting 3 hours or less. Factors etiologic to risk are related to the equipment, the perfusate, and technical execution of the procedure.

Existing equipment places blood in contact with varyingly antagonistic foreign surfaces and subjects it to unphysiologic flow conditions due to narrowed cannula and tubing sizes, angulation, and foaming. These combine to cause measurable destruction of all formed elements and alteration of blood proteins. Excessive postoperative bleeding may be observed and is generally associated with or the result of thrombocytopenia and increased fibrinolytic activity. A significant anemia may be noted 5 to 10 days postoperatively due to delayed red blood cell destruction. Although open heart surgery can be performed in specific cases without the need of donor blood, this has not yet become routinely practical in all cases. Serum hepatitis is perhaps the most common complication of extracorporeal circulation and is roughly proportional to the amount of blood (and therefore the number of donors) required. A postperfusion febrile syndrome with a hematologic pattern similar to infectious mononucleosis, probably caused by cytomegalovirus, has also been shown to be associated with infusion of large quantities of donor blood.

Certain complications are related to technical details of operative management. Air embolus results from failure to completely fill the pulmonary veins and left heart with blood prior to discontinuance of perfusion. Particulate emboli consisting of loose remnants of intracardiac thrombi or calcific fragments may be lethal, particularly if they lodge in the coronary or cerebral arteries. Arterial cannulae positioned in atherosclerotic femoral or iliac arteries may occasionally induce retrograde dissection of these vessels. When these arteries are extensively diseased, cannulation of the ascending arch of the aorta is advisable.

The postperfusion state tends to accompanied by measurable but tolerable alterations in organ function. Postoperative low-cardiac output is often of hypovolemic or bradyarrhythmic origin. Maintenance of proper intravascular volume is best achieved by using left or right atrial pressure measurements as guides to blood or colloid administration. Arrhythmias, particularly if associated with slow heart rates (50–70 per minute), are often effectively managed by rate augmentation employing atrial or ventricular pacing with wires implanted at operation. Discontinuance of digitalis preparations several days prior to surgery and avoidance of postperfusion hypokalemia will further reduce the incidence of postoperative arrhythmic problems.

Some degree of ventilatory and respiratory dysfunction is commonly observed, especially in patients who have been in pre-operative chronic left-heart failure or when perfusion has been lengthy. A combination of factors result in reduced diffusion of oxygen from the alveoli into the pulmonary capillaries. Elevated arterial carbon dioxide tensions in the early postoperative period are commonly the result of right-to-left shunting through atelectatic areas of the lung which are perfused but unventilated. Tracheal intubation and mechanical ventilation has been effective in management of these problems.

In the absence of blood incompatibility, postoperative renal failure consequent to perfusion per se is rare. It is probable that the hyperosmolar hemodilute perfusate used in most centers exerts a salutary effect in minimizing the incidence of this complication. When renal failure occurs (particularly acute tubular necrosis) it is generally observed in debilitated patients in cardiac failure who exhibit pre-operative nitrogen retention and a pattern of low cardiac output in the postoperative period. Precise intra-operative management and postoperative support and maintenance of cardiac output should reduce this complication to a minimum.

Emotional disturbances ranging from confusion, visual and auditory hallucinations to frank psychoses may develop in some patients following open-heart surgery. These are almost exclusively observed in adults and are believed to be caused by a combination of factors including perfusion trauma, pre-and postoperative apprehension, and lack of appropriate sleep patterns due to continuing activity in the intensive care unit. Constant reassurance and judicious sedation are required. Fortunately, the behavioral patterns tend to return to normal within 2 weeks of operation.

The Future of Extracorporeal Circulation

It is almost certain that further improvements in design of extracorporeal circulatory and gas-exchange systems will expand permissible periods of use to days or weeks, thereby making prolonged cardiac and respiratory assistance a practical reality. Ideally, such systems will not require donor priming blood or systemic heparinization of the patient. Thus, low-prime disposable pump-lung units with no direct gas-blood interface made of thromboresistant materials are required. There is urgent need for a non-toxic, non-antigenic plasma-like fluid capable of oxygen transport which can be used to prime extracorporeal circuits and replace blood lost during and after intracardiac and prolonged support procedures. Finally, the importance of pulsatile bloodflow requires further study since there is accumulating evidence that such a flow pattern enhances organ function under perfusion conditions.

Hemodynamic Effects of Cardiac Arrhythmias*

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Recent advances in intensive cardiac care and widespread use of continuous electrocardiographic monitoring have emphasized the frequency and nature of cardiac rhythm disturbances. Recognition, however, requires at least a consideration of therapy. Proper therapy is predicated on an understanding of the "natural history" and hemodynamic consequence of a specific arrhythmia.

The net effect of a particular arrhythmia is determined by the circulatory state of the patient as well as the nature of the rhythm disturbance. Usually "benign" rhythm disturbances such as bigeminy and atrial fibrillation, occurring in the patient with a limited cardiac reserve, may rapidly result in severe cardiac decompensation with myocardial ischemia, hypoxia, and hypotension, shock, or death. Furthermore, the arrhythmia may compromise the blood supply to the end-organs and thus produce myocardial infarction, renal failure, cerebrovascular accidents, hepatic necrosis, or infarction of the intestinal tract.

Since many arrhythmias are transient and cause only minor alterations of the circulation or occur in patients with less severely compromised circulations, the symptoms produced may be vague and nonspecific. Such symptoms include palpitations and episodes of weakness and fatigue; on the other hand, they may cause more serious symptoms and signs such as transient neurological deficits, lapses of memory, pre-syncope or syncope, increasing congestive heart failure, increasing angina, intermittent claudication, etc.

It must be remembered that the hemodynamic effects resulting from an arrhythmia are not due solely to the changes of cardiac function. The observed response of the circulation may well be due to peripheral effects. Thus, the status of the peripheral resistance, blood volume, baroreceptor activity, and venous return must be considered in any critical analysis of arrhythmic effects.

Rather than discussing the hemodynamics of particular arrhythmias, it is useful to consider the physiologic alterations that may be produced by any arrhythmia.

Heart Rate

The rate of contraction of the ventricles will determine the cardiac output if the volume of blood ejected with each stroke remains unchanged. Thus, rapid or slow rates critically affect hemodynamics. Bradycardia may produce profound effects, especially when the stroke volume cannot increase and peripheral compensatory mechanisms are inadequate. In many patients, however, the heart may compensate physiologically during slow heart rates by increasing stroke volume due to increased ventricular filling and ventricular wall pressure (Starling's Law). The net effect of increased stroke volume may compensate for a decreased heart rate resulting in insignificant changes in cardiac output which can be adequately compensated for by an increase in peripheral resistance.

In addition, bradyarrhythmias may permit the discharge of "irritable" pacemaker foci, thus predisposing to tachyarrhythmias and producing bradycardia-tachycardia syndromes.

If the heart rate increases beyond a critical rate (varying with the basic status of the cardiovascular system) the ventricle fills incompletely during diastole, resulting in a decreased output per beat. A similar, but transient, effect occurs during rapid irregular rhythms or multiple premature contractions (the earlier the contraction, the smaller the subsequent output).

"Atrial Kick"

Although appreciated by Harvey (1628), the importance of coördinated contractions of the atria and ventricles and the contribution of atrial systole to ventricular filling have recently been re-emphasized. In normal hearts atrial contraction may add between 10–20% of ventricular volume, whereas in severe valvular heart disease, such as mitral stenosis, the diastolic ventricular volume may increase over 50% during the period of atrial systole. It has been demonstrated that with an effective well placed atrial contraction a higher ventricular end-diastolic volume may be obtained with a lower mean atrial pressure than occurs when the atria are not functioning properly. Insufficiency of the AV valves may also be produced by the loss of coördinated atrial and ventricular contractions, resulting in detrimental cardiovascular effects.

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The hemodynamic effects of a rapid or slow heart beat may be minimal if the "atrial kick" is preserved, but when it is lost, the additional insult may cause decompensation.

Common examples of tachycardia associated with loss of coördinated atrial and ventricular contractions are atrial fibrillation, junctional (nodal) rhythm, and partial (second degree) heart block. The commonly used types of ventricular pacemakers should also be included, since atrial and ventricular contraction are not synchronized.

Method of Ventricular Activation

Several studies have shown that alteration of the normal sequence of activation of ventricular contraction results in adverse hemodynamic effects. Given two arrhythmias with identical coördination of atrial and ventricular contraction and the same heart rate, one demonstrating an abnormal sequence of ventricular activation (i.e., aberrant conduction) and the other normal sequence of activation, the former will result in greater alteration of hemodynamics. Such alterations, in addition to the absence of an effective "atrial kick", explain why ventricular tachycardia or junctional tachycardia with aberrant conduction is less well tolerated than atrial tachycardia. It may also explain why patients with sinus bradycardia may deteriorate rather than improve when the heart rate is increased by fixed-rate ventricular pacing (loss of "atrial kick" and normal ventricular activation).

Effects of Pharmacologic Agents

It should be mentioned that in the conversion of arrhythmias, especially tachycardias, to a normal sinus mechanism with drugs, the frequent myocardial depressant effect of these medications may result in a significant, although usually transient, fall in cardiac output. Thus, the administration of drugs such as lidocaine, quinidine, Procainamide (Pronestyl R), Diphenylhydantoin sodium (Dilantin R), and propranolol hydrochloride for management of arrhythmias should be performed under careful monitoring; the smallest effective dose should be used with the expectation that the patient may experience a temporary decrease in cardiac output even after normal rhythm has been established.

Conclusion

The clinical manifestations of cardiac arrhythmias may produce unusual or ill-defined symptom-complexes. The occurrence of incipient or increasing congestive heart failure, angina pectoris, intermittent claudication or episodic dizziness, fatigue, transient neurological disturbances, or paroxysms of dyspnea should alert the physician to consider the possibility of a cardiac arrhythmia.

It can be concluded that the hemodynamic effects of arrhythmias depend upon the underlying status of the myocardium, blood vessels, and end organs, the heart rate, preservation of the "atrial kick", and normal sequence of activation of the ventricles. The altered hemodynamic effects may be further aggravated by the effects of pharmacologic agents used to correct the arrhythmia.

Idiopathic Peritonitis in a Patient in Chronic Renal Failure*

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Whereas infection accounts for a high degree of morbidity and mortality in patients with chronic renal failure,[1] peritonitis is the primary factor for only a small percentage of infections. It is usually found in acute renal failure secondary to trauma;[2] as a complication of peritoneal dialysis;[3] following abdominal operations; or associated with pelvic inflammation.[4]

Described herein is, to the best of my knowledge, the first reported case of spontaneously occurring peritonitis in a patient with chronic renal failure.

Report of Case

A 52-year-old white male Capitol guard was brought to the Bethesda Naval Hospital because of weakness, fatigue, and hypertension. Approximately 15 years before, he had a sore throat which was followed by hematuria, proteinuria, edema, hypertension, and impaired renal function. A diagnosis of post-streptococcal glomerulonephritis was made, although no biopsy specimen was obtained. The patient was treated for approximately 11 years, and a gradual decline in renal function and persistent proteinuria were noted. The last recorded blood urea nitrogen (BUN) determination was 50 mg per 100 ml; creatinine, 5.5 mg per 100 ml; blood pressure, 160/100 mm Hg. After that time he was lost to follow-up for 4 years.

During a physical examination for employment the patient was found to have a diastolic blood pressure of 120 mm Hg and was referred to the Outpatient Clinic. He was edematous and appeared chronically ill. The BUN was 180 mg per 100 ml; creatinine, 18 mg per 100 ml; CO_2, 11 mEq per liter; potassium, 5.5 mEq per liter. Urinalysis showed 3+ proteinuria, microscopic hematuria, and numerous granular casts.

He was admitted to the hospital for evaluation of his renal status and for control of hypertension. He was lethargic and appeared chronically ill. The blood pressure continued to be 160/100 mm Hg. He was afebrile. Additional findings were a ventricular filling sound, and also shifting dullness and a fluid wave on abdominal examination. Repeat laboratory studies gave these results: BUN 200 mg per 100 ml; creatinine, 20 mg per 100 ml; CO_2, 8 mEq per liter; potassium, 5.7 mEq per liter. The hematocrit was 32%; peripheral white blood cell count was 6,800 with a normal differential; the platelet count was normal. A 24-hour specimen of urine gave 1.2 gm of protein and 72 mEq per liter of sodium. Endogenous creatinine clearance was calculated as 6 cc per minute. A culture of the urine gave no bacterial growth on 2 determinations, and blood cultures were sterile after 48 hours on 2 determinations.

Bed rest was prescribed, and the patient was treated with sodium, protein, and restriction of water intake, alpha methyldopa, and sodium bicarbonate. Forty-eight hours after admission the symptoms had not improved. He complained of pain in his right lower quadrant, which was tender. Also, asterixis was evident. He was still afebrile. The BUN at this time was 180 mg per 100 ml; the creatinine, 19.5 per 100 ml; the potassium, 4.9 mEq per liter; the CO_2, 16 mEq per liter; and the hematocrit was unchanged. The white blood cell count was 8,600 with a normal differential.

Because of the change in symptoms, peritoneal dialysis was instituted. A peritoneal catheter was inserted into the midline below the umbilicus with an Abbott Stylocath. After insertion of the Stylocath the trocar was removed and the catheter was positioned in the right lower quadrant, yielding purulent-appearing fluid with a protein content of 3.8 gm per 100 ml; sparce polymorphonuclear leukocytes; and gram-negative bacilli. Culture of the specimen gave *Escherichia coli*. Peritoneal dialysis was continued, and cephalothin, 1 gm intravenously every 6 hours and gentamycin in doses of 120 mg per day, were administered. Cephalothin was also added to the peritoneal dialysate.

In preparation for exploratory laparotomy the patient underwent hemodialysis for 6 hours on the 3rd hospital day. Sparse fibrinous material in the right lower quadrant and a

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normal-appearing biliary tree were found. Postoperatively he did well. The BUN stabilized at 80 mg per 100 ml and the creatinine at 12 mg per 100 ml. The patient was discharged after 3 weeks of hospitalization.

Discussion

In a recent review of the occurrence and types of infection in the presence of renal failure,[5] the authors commented that it was difficult to determine to what extent patients with renal failure are prone to peritonitis. Only 2 patients with chronic renal failure were reported with peritonitis as a significant infectious complication. However, their cases were either associated with a postoperative wound infection,[4] or were a complication of peritoneal dialysis.[3]

In our case, no history of preexisting abdominal disease was elicited nor any past abdominal operation. At laparotomy the gastrointestinal tract was found to be entirely within normal limits. Also, no previous peritoneal dialysis was performed nor was there evidence of generalized sepsis.

It has been demonstrated in the experimental animal that a peritoneal *E. coli* infection can occur after bilateral nephrectomy. The incidence of occurrence is decreased when the bacterial population of the intestine has been reduced by previous administration of an antibiotic. It was concluded that the gastrointestinal mucosa in the presence of uremia is especially permeable to bacterial migration.[6] Furthermore, patients with renal failure, for many reasons, have an altered inflammatory response[7] including impaired chemotaxis, impaired phagocytic activity of the polymorphonuclear leukocytes, suppression of reticuloendothelial function, and impaired antibody production.[8] These factors may account for many of the so-called silent infections in patients with uremia.

Our patient had long-standing chronic renal failure, and therefore it is likely that significant impairment of the host defenses to infection had occurred to account for the peritonitis. Also, lack of signs and symptoms are not inconsistent with other cases of severe bacterial infection in chronic renal failure.

Because infection in patients with renal failure may lead to somewhat rapid clinical and chemical deterioration, idiopathic peritonitis should be added to the list of causative factors of acute exacerbations of otherwise stable chronic renal failure.

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The Effect of Pyelonephritis on Renal Function A Survey of the 1970–1971 Literature

STEPHANIE M. YATER*

Many researchers in this field have found that urinary tract infection (UTI) affects urinary concentrating ability first and foremost. This is corroborated by the prevalent pathological findings in pyelonephritis, namely, tubular dilatation or atrophy and interstitial inflammation and fibrosis primarily in the medulla where urinary concentration takes place. In addition, the hypertonic medulla has been shown to be more susceptible to infection than the cortex. However, many workers have more recently found that certain other functional, and in many cases reversible, changes may occur.

In 1958 Winberg[1] demonstrated that a majority of children showed a decreased ability to concentrate urine for 3–6 weeks following acute UTI. This function is restored after therapy. It was hypothesized that damage to the collecting ducts and distal tubules was the most probable cause. Acidification capacity was found to be unimpaired, as was glomerular filtration rate (GFR). Thus, some damaged nephrons must be postulated to help maintain renal function in acute UTI, or early pyelonephritis, and glomerular tubular preservation must be inhomogeneous.

Mabeck's[2] studies in 1970 on bacteriuric women corroborated the finding that reduction of urinary concentrating ability is an early sign of pyelonephritis. He found that impairment of concentrating ability correlated with fever and flank pain and with an elevated coli antibody titer. He also showed that the recurrence rate was higher among patients in whom this function did not revert to normal.

In 1970 Kaye and Rocha[3], using rats, demonstrated that the rate of improvement in concentrating ability after experimentally-induced acute pyelonephritis correlated with the rate of decrease of bacterial titers in the kidneys. They showed that creatinine clearances were not affected, but that histological findings in the kidneys correlated poorly with the urinary concentrating ability. Since this defect is rapidly reversible, it must be due to transient damage rather than to actual nephron destruction. These authors think that the explanation may be either changes in permeability of the collecting tubules and/or decreased osmolality of the medullary interstitium due to a decrease in urea concentration. They hypothesize that these defects could be caused by products of bacterial metabolism or inflammation either affecting permeability directly or inhibiting necessary enzymes.

Berg et al[4] in 1971 found that reduction in reabsorption of bicarbonate may be one of the earliest signs of pyelonephritis. This function was found to be affected before the filtration rate was reduced, because some of the patients studied who had normal GFR's were found to have reduced bicarbonate thresholds. These patients, who nevertheless have normal distal hydrogen ion secretion, are able to lower their urine pH once this threshold is passed. Thus it can be postulated that the glomerular tubular balance for bicarbonate reabsorption is disturbed and that the nephrons do not function homogeneously in this regard. Histochemical studies have shown reduced carbonic anhydrase activity in some cases.

In 1968 Calcagno et al[5] found that renal blood flow is reduced before reduction of any other functional parameter in infants and children with UTI's. They suggest that post-glomerular capillaries and proximal tubules in the cortex, where renal blood flow is highest, may be rendered ischemic by the inflammatory process.

In patients with chronic and recurrent UTI's, and with renal parenchymal changes demonstrated by IVP as well, disturbances of additional renal functional parameters were noted by Aperia et al[6] in 1971 who found reduction of GFR to be the major lesion. These workers studied renal function during the transition from hydropenia to water diuresis. In 3 of 11 patients with renal parenchymal changes and reduced GFR's, they found an increase in fractional sodium excretion during hydropenia. However, the fractional sodium excretion during the transition from hydropenia to water diuresis increased almost as significantly as the controls who had normal GFR's. Thus glomerular tubular balance must be reset at a new level in order to maintain fluid and sodium homeostasis in the face of a reduced number of nephrons. A natriuretic factor has been postulated to effect this adjustment during low GFR's. These same patients also showed a reduced concentrating ability, again demonstrating reduced sodium reabsorption in Henle's loop.

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In chronic pyelonephritis impaired concentrating capacity is thought to be due to a reduction of functioning nephrons and thus a higher solute load per nephron. However, the degree of impairment of concentrating ability is greater than can be explained by this hypothesis, and one must also presume perhaps disruption of medullary hypertonicity (decreased accumulation of urea has been hypothesized) and/or diminished sensitivity of the collecting tubules to ADH. Five patients in this study could not increase their diluting capacity during the transition from hydropenia to water diuresis, suggesting diminished distal sodium reabsorption following a water load.

Aperia et al in 1971[7] studied control of sodium homeostasis in children with recurrent UTI's by means of oral and intravenous sodium loading. They found that patients with low GFR's were unable to respond rapidly to the increased quantity of sodium, probably by inability to decrease tubular sodium reabsorption. During transition from hydropenia to water diuresis the extracellular volume expansion inhibited sodium and water reabsorption almost as significantly in patients with low as with normal GFR's, as noted above. But with a superimposed sodium load the patients with low GFR's were unable to further decrease sodium reabsorption. It was postulated that this might be due to the already maximal stimulation of a "factor" which controls sodium reabsorption.

In 1971 Berg et al[4] studied maximal ammonia excretion in patients with recurrent UTI's following an oral ammonium chloride load. They found that in patients with low GFR's the total ammonia excretion was reduced but was normal when calculated per functioning nephron. Berg in 1971[8] found that 5 of 9 patients with normal GFR's were unable to acidify the urine adequately and postulates that defective renal acidifying capacity might be an early sign of renal damage.

In 1958 Lathem[9] reported on four patients with chronic pyelonephritis who had hyperchloremic acidosis, hyperkalemia, and acid urine. These disturbances were attributed to damaged tubular function with (since the patients were not azotemic) normal glomerular function. It was postulated that the tubular secretion of potassium and hydrogen ions were impaired, and a greater impairment of ammonium than of titratable acid excretion was proposed.

Since the bicarbonate concentration of the blood is reduced, hyperchloremia develops in order to maintain electroneutrality. However, when renal disease becomes chronic and affects the glomeruli as well as the tubules, the concentration of undetermined anions increase and the chloride concentration will then revert toward normal. Thus, hyperchloremia may be found only in early pyelonephritis.

In 1970 Vagnucci[10] reported on a patient who had chronic pyelonephritis with severe hyperkalemia and diminished aldosterone production, while other adrenal hormone levels remained normal. Aldosterone production did not increase under sodium restriction, intravenous ACTH, or angiotensin II. DOCA therapy corrected the hyperkalemia, and a defect of C 18 hydroxylation was postulated. Since this patient evidently had no signs or symptoms of aldosterone deficiency previously, perhaps his pyelonephritis triggered the uncovering of the postulated deficiency.

In a study on children with pyelonephritis[7] adrenal steroids were measured and found to be normal (sometimes even elevated in the case of aldosterone) in all cases. Selective aldosterone deficiency accompanying this disease has been reported mainly in adults.

Since many parameters of renal function are affected during the course of urinary tract infection and can be used in its diagnosis and response to therapy, those which are the least traumatic for the patient should be chosen.

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Traumatic Coronary Arterial-Right Ventricular Fistula[*]

Report of a Case

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ANTONIO C. DELEON, JR., M.D.[||] AND WILLIAM L. JAMISON, M.D.[¶]

Arteriocameral fistulas caused by penetrating injuries of the heart are rare.[1-6] We recently studied a patient with a continuous murmur due to a left anterior descending coronary artery to right ventricular fistula following a shotgun injury to the chest.

Report of Case

F. S. (GUH #222410), a 37-year-old man, was in good health until October 1970 when he was accidentally hit in the anterior chest with #6 birdshot while squirrel hunting. Several of the shot were surgically removed from the chest wall. In December 1970 a continuous precordial murmur was first detected, but the man remained asymptomatic.

The blood pressure was 118/74 mmHg and the heart rate 72 beats per minute. The peripheral arterial pulses and the jugular venous pulse were normal. The only abnormality in the cardiovascular physical examination was a grade 3/6 high-frequency continuous murmur localized in the 4th and 5th intercostal spaces to the left of the sternum (Fig. 1).

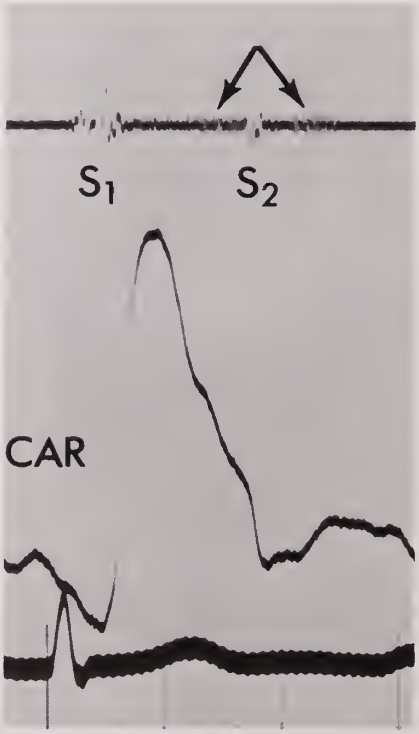

FIG. 1. Phonocardiogram from the fourth intercostal space along left sternal border. Arrows point to the high-frequency continuous murmur. S_1, S_2–first and second heart sounds. CAR–carotid arterial pulse.

The electrocardiogram was normal. Chest roentgenogram was normal except for scattered radiodensities of the pellets, 1 of which at fluroscopy was intracardiac (Fig. 2).

Cardiac catheterization (2-2-71) recorded normal intracardiac pressures. Hydrogen inhalation detected a left-to-right shunt localized to the right ventricle. Selective coronary cineangiocardiography demonstrated normal left circumflex and right coronary arteries but a dilated left anterior descending coronary artery with a direct communication to the right ventricle (Fig. 3.). The left-to-right shunt was too small to be quantified by Krypton 85 inhalation.

Discussion

Continuous precordial murmurs following penetrating injuries to the chest can be due to communications between an internal mammary or intercostal artery and accompanying vein,[7] an extramural coronary artery and its accompanying vein,[8] or to communications between aortic root and right heart,[9,10] aortic root and pulmonary trunk,[10] right coronary artery and right atrium[1-6] or right ventricle,[2] or as in this case left coronary artery and right heart.

The murmur of traumatic coronary arterial fistula has an onset varying from shortly after injury, as in our patient, to considerably later.[6] In 1

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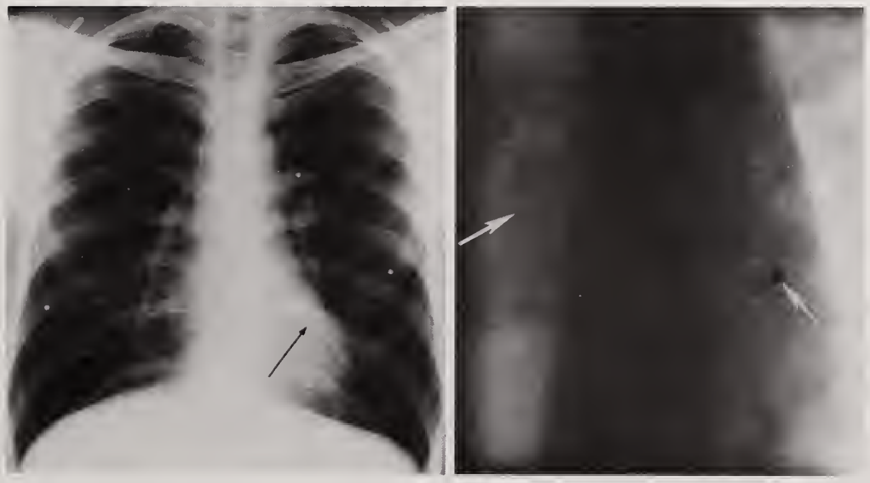

FIG. 2. Left panel–X-ray showing many shot-gun pellets. Arrow identifies the intracardiac pellet faintly seen. Right panel–Frontal projection following left coronary arterial injection. Left arrow points to contrast material in outflow tract of right ventricle. Right arrow points to the intramyocardial pellet identified by black arrow in the plain film of the chest.

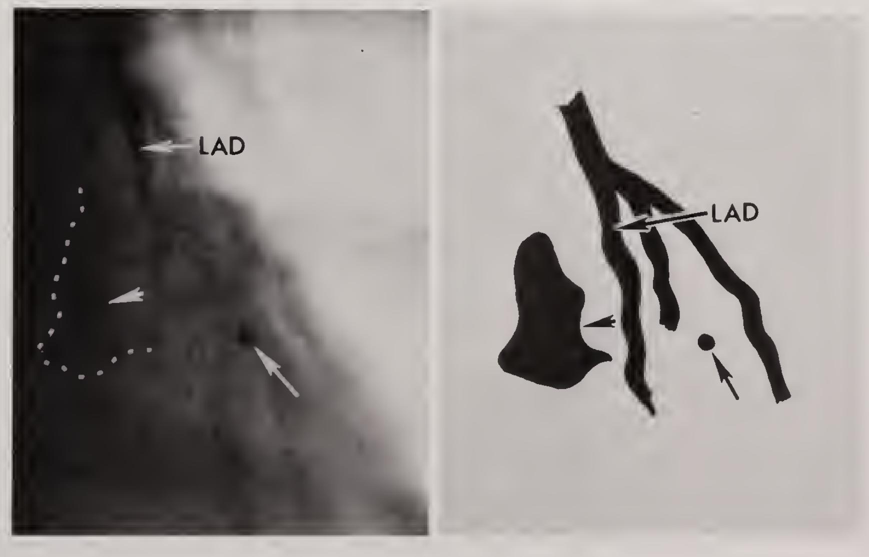

FIG. 3. Left panel–Left coronary arterial injection in left oblique postion. Upper arrow points to left anterior descending coronary artery (LAD). Dotted area and middle arrow identify contrast material in right ventricle. Lower arrow points to shot-gun pellet. Right panel–Sketch of features described above.

instance the original injury caused a false aneurysm of the right coronary artery which ruptured into the right atrium 2 years later.[6] A recent report[11] documented a shot-gun-pellet-induced left anterior descending coronary arterial aneurysm that remained stable angiographically over a 6-month period. These observations strengthen the hypothesis that traumatic coronary arterial aneurysm with subsequent rupture is a cause of the delayed development of some coronary arteriocameral fistulas.

The size of the communication governs the amount of augmented coronary arterial flow and left-to-right shunting. Increased flow acting over a long period of time may convert the involved coronary artery into a dilated, tortuous vessel[3,12] similar to that seen in congenital coronary arterial fistulas. Because our patient had a very small shunt and no symptoms, we elected to defer operation despite susceptibility of the fistula to infective endocarditis. However, several patients with traumatic arteriocameral fistulas have developed congestive heart failure and have undergone operation with varying success.[2,4,6]

Summary

This report documents the rare occurrence of a traumatic coronary arterial–right ventricular fistula following an accidental shot-gun injury to the front of the chest. The causes of continuous murmurs due to penetrating chest injuries have been reviewed and the literature relevant to traumatic coronary arterial fistulas briefly summarized.

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Anemia, Briefly Discussed*

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Anemia is usually symptomatic of underlying disease, not an entity in itself. Evaluation of anemia may help diagnose and treat a patient's major disease. How thoroughly one studies anemia depends on its severity, its cause, and the patient's other diseases. The more severe the anemia, the more complete should be the initial evaluation. Moderate iron deficiency anemia may be easy to detect and treat; extensive blood and bone marrow studies are not needed. The patient with chronic renal disease and severe azotemia needs little study for his hematocrit value of 25 (hemoglobin 8.2 grams), since they are probably related. If prompt transfusion is considered essential, a more complete initial work-up should be done than if no immediate hematologic therapy is demanded. By analysis of the patient's data base and problem list, one should always attempt to assign a pathogenesis or an etiology to anemia.

A classification of anemia based upon erythrokinetics and pathobiochemistry is shown in Table 1. A key laboratory parameter is the reticulocyte count, which is best expressed in absolute terms (% reticulocytes X RBC; normal = 60 – 80,000) and often must be done serially. The "response pattern" can and must be distinguished from that of chronic red cell loss (hemolysis or bleeding, figure 1), even if initial observations are missing. With anemia, reticulocytes are released earlier from the bone marrow, remaining longer in the peripheral blood before losing their reticulum to become erythrocytes. With a correction for this longer sojurn in the circulation, one has a reliable quantitative measure of erythropoiesis (divide reticulocytes by 1.5 for Hct = 35%; 2.0 for Hct = 25%; and 2.5 for Hct = 15%). Red cell morphology and indices (MCV, MCH, and MCHC) may be a useful guide to pathobiochemistry. Small red cells with or without hypochromia signify poor hemoglobin synthesis, most often from iron deficiency. Anisocytosis is greatest with a maturation defect; oval macrocytes suggest a nuclear maturation defect. Increased numbers of red cells with fine punctate or diffuse basophilia indicate increase red cell production. Spherocytes suggest hemolysis. Other morphologic features (target cells, sickled cells, red cell fragments, etc.) may also be helpful diagnostically. Bone marrow examination may not be necessary; when needed it is best obtained as part of a consultation covering the entire hematologic problem rather than as an isolated tissue examination.

The patient's age and sex is often helpful in managing anemia. For example, in infancy, childhood or adolescence, anemia is most likely caused by either iron deficiency or infection. In these age groups, the iron deficiency is not commonly the result of chronic bleeding. Enlarging body size requires an enlarged red cell mass which requires iron. Iron deficiency is also common in adolescent and premenopausal adult females because their diet may be unable to furnish the increased needs from growth, pregnancy, or abnormal (even sometimes normal) menstrual blood loss. A therapeutic trial with iron as the only hematinic confirms the diagnosis when follow-up studies indicate improvement and an ultimate return to normal. Elective gynecologic surgery probably should not be performed until after such anbulatory therapy restores the blood to normal or nearly so. Failure to respond demands an explanation. In other groups of patients, iron deficiency anemia should always provoke a search for occult bleeding and its cause. Even within these groups occult bleeding may be signaled by recurrent or unusually severe anemia.

Only in alcoholism or pregnancy is folate deficiency at all likely in the United States. Deficiency of vitamin B-12 is even less common. Except for these two vitamins and iron, nutritional deficiency rarely causes anemia. Hence "nutritional anemia" is an obsolete and possibly misleading diagnosis.

Acute episodic anemia usually comes from bleeding or less commonly hemolysis. Until the blood loss results in iron deficiency, these may be very difficult to differentiate, especially since 400 ml or more of blood may ooze into a day's stool without a change in color. Episodic hemolysis

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TABLE 1: Simplified Classification of Anemia Based Upon Patho-Biochemistry and Physiology

	Hypoproliferative	Maturation Defects	Hyperproliferative
Reticulocytes	Decreased	Normal or decreased	Increased
E/M (marrow)*	Normal or decreased	Increased or normal	Increased
RBC (smear and indices)	Normal cells uniform	Anisocytosis Small cells or large cells pale cells or normal hemoglobin	Fine punctate or diffuse basophilia increased Specific abnormalities (sickled cells, spherocytes, fragmented cells, etc.)
Etiology or pathogenesis	Hypoplastic anemia Most secondary anemias (infection, renal disease, etc.)	Cytoplasmic maturation defect (iron deficiency thalassemia, etc.) Nuclear maturation defects (B-12 or folate deficiency, etc.)	Bleeding Hemolysis Hereditary Acquired

Only the more common diagnostic terms are included at the bottom as examples where needed. (* E/M = erythroid to myeloid ratio in the bone marrow.)

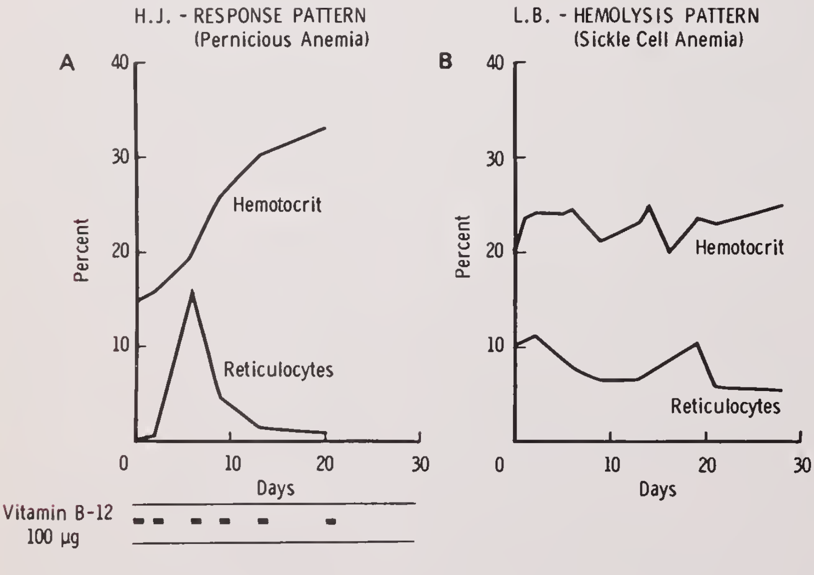

Fig. 1A: Pattern of hyperproliferative anemia from hemolysis or bleeding. Note fluctuating elevation of reticulocytes (increased red cell production) in a steady state (relatively constant anemia) = increased red cell loss (hemolysis or bleeding). 1B: "Response Pattern" of hematocrit and reticulocytes. The peak reticulocyte count comes between 5 and 9 days after the stimulus. Stimuli are many and varied: start of therapy as with iron, B-12, or folic acid (as in this instance); sudden red cell loss as by an episode of bleeding or hemolysis; half reversible marrow depression as with chloramphenicol (sometimes); etc.

may be caused by drugs or infections and often is accompanied by the transient presence of red cell Heinz bodies and a brief elevation of indirect bilirubin. The transient nature of Heinz bodies, indirect hyperbilirubinemia, and even a positive test for fecal occult blood needs emphasis, because they can readily be missed through delay.

Chronic hemolysis may be hereditary or acquired. Hereditary disorders are most common in certain ethnic groups, such as sickle cell disease in Afro-Americans. Specimens to detect abnormal hemoglobins by electrophoresis obtained after a blood transfusion are useless. Indirect hyperbilirubinemia or jaundice without bile in the urine usually means hemolysis. Other laboratory tools may be helpful but should be ordered judiciously rather than as part of a "shot gun" anemia work-up.

The goals of the patient's primary physician should be: (1) to make a proper diagnosis of anemia and apply specific therapy if possible; (2) if not, to obtain those clinical and laboratory data that are transient in nature or irretrievably altered by therapy (especially by blood transfusion); (3) to avoid therapeutic confusion by shot gun or injudicious treatment; and (4) to request suitable consultation when needed.

The Case of the Defrocked Nurse*

ZIGMOND M. LEBENSOHN, M.D.†

Let me start by saying that I like nurses—frocked or defrocked. Ordinarily I have no trouble writing about them. But this article—for reasons which may soon become apparent—has given me all kinds of problems. The idea of writing a piece on the subject of nurses without uniforms has been incubating in my mind since I first began noticing defrocked nurses working on the wards of psychiatric hospitals in various parts of the country. My thoughts gradually took form. All I needed was a title.

My first working title was "A Nurse is a Nurse is a Nurse" in order to emphasize the obvious Steinian truism, uniform or no uniform. Then, lest the main point be missed, I added an alternative title which read "Uniform, Uniform, Who's Got the Uniform"? By this time I was beginning to get carried away by the philosophic implications of blurred categories and, leaning heavily on Kant, I came up with my third title, "The Categorical Subjunctive." Finally, to nail down the role identity problem, I added "You Can't Tell the Players Without a Program." It was now obvious that none of these four titles could stand by itself. But it was equally obvious that no editor would stand for all four of them strung together however admirable they might serve as a mood setter. Hence, my relatively brief title and my tediously long explanation. Now to the substance of my plaint.

Blurring the distinctions between long cherished and formerly neat categories has become an obsessional preoccupation of our troubled times. The formerly sharp cleavages between student and teacher, between laborer and capitalist, and even between male and female are no longer as clear as they used to be. Egalitarianism and unisex are the dominant themes of the moment.

The wards of the mental hospital, and even the psychiatric units of the general hospital, have not escaped these "homogenizing" influences. The current vogue (and let us hope it is only a vogue) of dispensing with the traditional white nurse's uniform in psychiatric hospitals has penetrated many previously staid psychiatric establishments. Militant members of the new "nurses' lib" have pressed psychiatric directors to win freedom from the uniform. When the director is kindly, experimental, uncertain, or weak, the nurses win their point. When the director stays firm, he is accused of being "some kind of fascist reactionary or male chauvinist pig." The visitor to a ward which has succumbed to the no-uniform craze is hard put to distinguish between patients and staff. This delights the proponents of the "new look" because this is precisely what they want.

Let us examine the issues. The champions of the no-uniform school self-confidently claim that they are acting in the cause of "progress," that they are deliberately removing the distasteful uniform which, in their minds, has become the hated symbol of authoritarianism in a subculture where authority is evil. The uniform, they contend, is a symbol in whose name patients were made to feel inferior—a symbol now despised because some of those who wore the uniform abused the authority which came with it—a symbol of establishmentarianism in a time of strong anti-establishment feelings. If we question further, we are told that the role of the psychiatric nurse has changed from the traditional bedside comforter to one in which she enters actively into therapy, administers the nursing unit, leads group sessions, and, in general, interacts with patients on a person-to-person level. In such a role many nurses feel that the white uniform acts as a barrier to communication and to the establishment of rapport. Also, they note that the psychiatrist, still at the top of the pecking order, almost always appears in civilian dress—ergo, what is good for the psychiatrist must now be considered good for the rest of the professional staff. Somehow or other the word got around that if everyone on the ward was just "one happy family" without distinguishing uniforms or badges, patients would feel more comfortable, and more meaningful therapy could take place.

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We have long suspected that many hospital practices are established not so much for the benefit of the patient but rather for the convenience and comfort of the staff. Arbitrary meal hours and visiting hours are only two examples. The abolition of nurses' uniforms falls into the same category. Why buy uniforms? Why bother to change into street clothes when taking patients for a walk or an outing? Why indeed?

Another explanation for the phenomenon may be found in the very nature of psychiatry itself, its multiplicity of treatment methods, and the enormous difficulty in assessing their effectiveness. From the historical perspective our treatment of the mentally ill has shown far less fundamental change over the centuries than have medicine and surgery. Aside from the relatively recent introduction of somatic therapies and psychotropic drugs, psychiatric treatment has remained essentially unchanged. Suggestion, reeducation, catharsis, hydrotherapy, milieu therapy, moral suasion, and many other approaches that we still use in psychiatry were also practiced by the ancient Greeks. Psychiatry has never developed its own "penicillin." Thus, psychiatry, envious of the fantastic advances in medicine and surgery, was hard-pressed to keep pace. Unable to achieve similar breakthroughs, psychiatry had to search hard to find what could be changed. A scapegoat was found in the form of the ancient and honorable medical model which had served psychiatry so faithfully for centuries. At first it was looked upon with suspicion. Later it became quite fashionable in many quarters to reject it outright. The further away we drifted from medicine the more important it was to divorce ourselves from the traditional symbols of medicine. White smocked psychiatrists who used to work in hospitals deserted the smocks for mufti and the comfortable isolation on the consulting room. And nurses discarded their uniforms for the professional anonymity of civilian dress. Such changes, for the most part, I suspect, are made primarily for the sake of change. Since the move to abandon the uniform has not as yet been accompanied by a therapeutic millenium, one suspects that the movement is based more on superficial fashion than on profound insight.

Movements to abolish uniforms and class distinctions have appeared periodically throughout the course of history. In times of great unrest and revolt there is a great craving to throw off the shackles which cramp the free spirit. The uniform becomes a perfect symbol of these shackles and out it goes! But the experience in both the French Revolution of 1789 and the Russian Revolution of 1917 showed that such movements were short-lived. Even the Soviets, who tried to remove all class distinctions and uniforms in their universities, schools, and even in the military, had to reinstate the uniform to avoid chaos. There appears to be a basic human need for structure in society even though excessive emphasis on structure can be stifling. As in most societies, a happy balance between structure and freedom also works best for the microsociety of the psychiatric hospital.

For some nurses, working without a uniform has become a status symbol and an inverted form of snobbism, similar to the British custom of addressing a surgeon as "Mister." Although the custom has different historical roots, there are certain important similarities. The British surgeon zealously eschews the distinguishing title of "Doctor" as if to indicate that he does not need a title to set him apart from laymen. Similarly, the nurses feels that she does not need the distinguishing white uniform to set herself apart from the patients.

In their enthusiasm to get on the bandwagon, proponents of the no-uniform movement may have lost sight of the subtle but powerful advantages of the uniform. Aside from establishing immediate role identity to the new or confused patient, the uniform is highly effective in maintaining "social distance" (to use Harry Stack Sullivan's phrase) between patient and staff. Anyone who has had experience working in a psychiatric hospital knows of the explosive potential for runaway transferences which can develop between a patient and a vulnerable staff member. I use the term "staff" to include psychiatrists, nurses, technicians, aides, and, in fact, all personnel working closely with patients. Although no group is immune, it is the nurse and her support staff who spend the longest hours interacting with patients and are therefore exposed to the greatest risk of emotional over-involvement. Nurses who choose civilian dress in order to enhance rapport may not always realize that their seductive potential may also be enhanced by the same maneuver. There are always certain patients who are vulnerable to over-involvement. Removing the conventional barrier of the uniform makes them even more vulnerable. The white uniform is a quiet reminder that the warm, therapeutic relationship has its limits.

The ideal psychiatric hospital must also set limits for its patients but it must not become a prison; it should have a comfortable, home-like atmosphere but must not be so comfortable that it becomes a substitute for home. We must never forget that the basic purpose of a hospital is to receive sick people, treat them, and return them to their homes, if possible, as quickly and safely as we can. In this spirit, the nurses' uniform is a great advantage in gently reminding the patient of the temporary nature of his stay. It silently tells him that the relationships he makes during his hospital stay, important as they may be, are no substitute for the relationships in his extramural life.

Let us examine what happens to the patient who is admitted to one of the newer psychiatric units where the nurses are not set apart by the traditional white uniform. Already confused by his illness, distressed by the events leading to his admission, the patient is further confused by being unable to distinguish quickly between nurse, aide, or patient. Even if he is introduced to some of the staff, it will take him days or weeks to distinguish staff from patients. When a patient is confused, it is a therapeutic axiom (medicalese for common sense) that one should make things as unambiguous as possible. The uniformless ward worsens ambiguity instead of lessening it.

Enthusiastic adherents of the new school act as if they are ashamed to wear the traditional white uniform of their profession. One wonders what they are really ashamed of. Nursing has a rich and noble tradition and the white uniform is the symbol of the helping hand to the sick. Why not use it to its fullest in the treatment process?

Perhaps the confusion which spawned this practice in the first place goes deeper than appears on the surface. Perhaps it is the confusion and chaos which comes when psychiatrists, nurses, and hospitals desert the medical model and embrace one of the newer models such as the moral model, the family model, or the social model. In such models there is, indeed, little distinction between patient and healer, between the sick role and the well role, between nursed and nurse. Abolition of the uniform is only an outward manifestation of what has already occurred inside. The Aesculapian role, once so highly prized, is abandoned by these new adventurers and, with it, all of its trappings and traditional authority. Of course, there is not a shred of evidence to suggest that the new system cures people faster, more permanently, or more effectively. But the acolytes of the new order shrug off such questions in much the same condescending manner in which the true believer treats the doubting questions of the infidel.

Although the no-uniform practice is mischievous enough in the psychiatric hospital, it is particularly unfortunate in the psychiatric unit of the general hospital. All of the architectural and administrative details of the unit are usually designed to diminish the sense of "differentness," strangeness, and alienation. Every attempt is made to make the unit look and operate like other services in the hospital. Abolition of the uniform emphasizes the differences. Nurses on all other units wear white uniforms—but not on psychiatry! No, indeed! Patients there are "special" and need specialized treatment.

I strongly suspect that the non-uniform trend will eventually run its course. Even so, many hospitals will continue the experiment but, in the final analysis, it is the person behind the uniform that matters. A good nurse is rendered not less effective but more effective by her uniform. It stands to reason that a sick person will accept medication and treatment much more readily from a nurse in uniform than from someone in street clothes who must identify herself each time she meets a new patient. The sooner psychiatrists go back to the medical model and begin to treat sick people in their traditional role as physicians, the sooner will the psychiatric nurse reclaim her uniform as a useful and unambiguous symbol of help to sick people.

The Future of Private Medicine*

C. WILLARD CAMALIER, JR., M.D.†

At times a single word can make a big difference and this is one of these times. The title assigned me is the Future of Private Medicine. Had it been the future of the private *practice* of medicine there would indeed have been a problem for a definition of this I do not have. However, private medicine is more easily seen as any type of medicine which is not carried out and supplied by employees of the government whether it be Federal, State or county government involved.

Within the broad category of non-governmental medicine it must be recognized there will exist a multiplicity of methods or arrangements whereby medical care is delivered to the patient. This is the pluralistic approach so enunciated and discussed by the A.M.A. a number of years ago when it pointed out that different circumstances, different geographic areas, different cultures, and different economic factors can all require different means of caring for people. Before getting into possible changes contemplated by various planners—some well motivated and some put forward purely because of popular appeal for political purposes—these latter usually called womb to tomb health services—a few facts are in order. These may be of interest and unless otherwise indicated are as of December 31, 1970.

As of that date there were 334,028 physicians in the United States. Of this number 310,845 were active M.D.s and 278,535 were involved in patient care. This was 83.4 per cent of all physicians. 6.9 per cent were inactive or could not be located anywhere. As a primary activity medical teaching took 1.7 per cent, administration 3.6 per cent, research 3.6 per cent, and miscellaneous 0.8 per cent. Therefore, in 1970 there was one M.D. for every 632 patients as contrasted to 1960 when there were 712 patients for each physician. In line with multiple approach to care in 1969 there were 40,093 physicians in 6,371 group practices and a total of 7,144 hospitals in the private field offering 1,649,663 beds and of these hospitals 5,853 of them were community hospitals. The average work load of physicians in private medicine during 1969—the last year surveyed-showed an average week to be 120.1 total visits for all fields of medicine while the general practitioner or family physician had the highest load of 159.5 visits per week. Of the roughly 289,000 physicians in patient care 8.4 per cent are employed by the Federal government, therefore, it is seen there is an amazing 91.6 per cent of physicians in patient care in the private field. It is again of interest to note that while in 1950 there was one physician to each 711 patients, there was one to each 712 in 1960, but in 1970 there is one physician to each 632 patients. The last decade then has produced more M.D. growth than population growth on a percentage basis. As one looks at the factors which may affect the private medicine picture look again at the group practice figures which involve 40,093 physicians. Of these groups, 49.7 per cent are found to be single specialty—12.3 per cent are groups of general practitioners while 38 per cent are multi-specialty groups.

Having looked at a portion of the manpower available in the private sector of medicine (total bodies including physicians in the total health field is not fully known, but is somewhere around 3.7 million persons) and recognizing the total health insurance benefits payments in the last year studied—1969—were 14 billion and 267 millions dollars while medicare for that year for personal health care expenditures amounted to 6.3 billion and medicaid for 2.175 billion dollars, it is time to take a look at what may be in the future and why consideration is being given to so many schemes.

It is my intention to stress in the beginning that the "pitch" of all proponents of change in delivering health care is that there will exist two things: (1) easy accessibility to complete or comprehensive care to all people wherever they live and (2) this can be done more cheaply and more fully by government and its funds either federal or federal-state combination. One has only to look at the broken promises and financial failure of the Title 18 and 19 programs (known as Medicare and Medicaid) to see that such ill-conceived programs can not deliver the politicians promises. Once again the M.D. is being asked—or rather told—if he takes care of these medicare people he must be content with 1968-1969 fees and with the 75th percentile of this. No consideration is given to the matter of the rise in expenses or cost of living-once more the physician is underwriting a federal program which is on poor financial grounds. The Federal Government or indeed the state government has not yet been able to do anything as cheaply as the private sector in any field. The second item of accessibility is a subject to be discussed.

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For a long time the popular appeal for support has been the promise of easy accessibility of medical care to everyone in the U.S., yet those who promise this and put this forward have several approaches they would like to use and herein lies the future of medicine as we know it in all its forms. First, the manpower side and then the financial which are interlocking but which I will try to separate. I wish to point out that although the detractors of private medicine try to link together all medical costs and put that "monkey" on the physicians' back there are honest people who know and see and say the facts namely that 70 per cent of the medical dollar expense is for hospital care and about 25.4 per cent for physicians' services. The rest goes for drugs, ambulances, etc. The physician is in no position to handle hospital costs and his only contribution here can come on such drugs, tests, etc. which while ordered by him show up on the hospital bill. These however can not be labelled hospital costs-they are not related to the room rate, the labor payroll, the cost of soap, linen, germicide, pens, paper, etc.-even if they do appear on the bill.

What about the physicians of this country and how are they nationwide looking at the situation. There are a number of physicians who are leaving, whenever possible, solo practice in favor of full-time employment by industry, hospitals, or groups and they are doing this under the influence of several factors: (1) Either they are close to 60 years or over and are tired of the paper work involved in claim forms for insurance, Blue Shield, or Medicare or Medicaid or Champus or Workmen's Compensation, etc. or they are younger and want what all of industry has—a 40 hour week—paid vacations-paid sick leave and a pension or retirement plan at the end of the road. The attraction of only working for 8 hours and then being free, when added to the growing disdain the public has been brainwashed into, makes such physicians refuse to be pawns. (2) The continued rise in professional liability insurance premiums with the continued "sitting duck" position of a physician in relation to an unscrupulous lawyer has led many who can afford it to leave medicine altogether and do something else. Yet what we face in this country is not a true shortage of physicians as much as we do a maldistribution. The doctors are not in the rural areas or in the slums of our cities. While access to the health care is available it often takes some work to get there. We come at this point to a very definite point of principle—it is—shall the physicians of this country when they graduate from medical school be free to choose their field of work or shall they be told where to go and what to do? The only one with the power to do this if it were not considered unconstitutional (at least today) is the Federal government. So private medicine has a potential threat from government whereby decisions of the future of the graduates or those in practice may not be theirs to make. Private medicine may soon be standardized again by Federal government. Congress is now considering new Social Security amendments which again will contain the language known as the Bennett Amendment on the Senate side. This would create a PSRO or professional services review organization. While this set-up under rules and regulations of the Secretary of HEW is at the moment aimed only at medicare and medicaid programs it can easily be extended to any program where government funds are involved. A PSRO is an organization to do continuing audit of practice methods of office, home, and hospital care, of drugs used, of tests done and the necessity of doing them, of surgery done, etc. which by the contemplated language cannot be a medical society and may even be a non-medical group. This is not peer review and many physicians will not subject themselves to such control by lay persons especially where the PSRO standardizes care without thought of anything except the cost reduction factor. And yet a physician will not go into slum or rural areas of certified medical need because of numerous factors. He is lacking contacts with other physicians, he is often without a decent life for his family, and his inability to earn a living. Even where governmental funds are involved in paying for care this physician is subject to harassment by Social Security and Internal Revenue Service when his incomes involving this Federal money reaches an arbitrary $25,000 gross in any year or portion thereof. No matter how little he charges or how many hours he works—the arbitrary figure is reached—his future payments stop—his office is invaded and may be padlocked—his home may be searched—his records may be seized—that is the type of thing which can and does drive the physician away from contact with or caring for any persons of these programs.

To turn to the financial side for a few moments. As Federal dollars become more and more involved in the health care picture obviously this money must be well spent. Federal money, exclusive of hospital or Hill-Burton funds is found directly or indirectly in Champus program in the Federal Employees Health Benefit Program where a percentage of the premium is paid federally, in Medicare, in Medicaid, in certain agencies of the government, in welfare under matching state-federal funds, in grants to hospitals and medical schools, in loans to schools and students—or to put it another way in 1970 the Federal government spent a total of $16.7 billion for health care. It is interested yet it cannot honestly believe it can do it more cheaply especially as against all independent figures both the Senate Finance Committee and House Ways and Means chose to believe its actuaries on the cost of Medicare and Medicaid. As we know, they were so wrong. It is pathetic as taxes go up and up trying to make both of these viable programs. It is the pet idea of innumerable persons in a government that one can cut the cost of health care by bringing all people into a maintenance program where like an automobile they will be recalled at intervals and necessary parts checked thereby insuring good health at a low cost. Under-Secretary Veneman of HEW on March 23, 1970, first used this idea of an HMO or Health Maintenance Organization and while nobody fully has a definition it is being popularized in the news and is glibly used in Congress and elsewhere. Yet it is an old concept—it is a corporation which contracts to furnish comprehensive medical care including hospitalization, ambulatory care, drugs, physicians' services, etc. on an annual pre-paid per-capita basis to persons enrolled in it. Physicians' services are provided by MDs who are either partners or employees or they may be, as in the case of the Permanente organization, a group which is reimbursed on a per-capita basis while within the group fee-for-service charges may be the basis used. The basic idea is that if a fixed annual payment is available regardless of the volume of services rendered there is a financial incentive (in the form of a year-end bonus) to control costs and provide only the least expensive service required appropriate to the enrollees needs. While the claims are made that there is a reduction in costs no figures from any place have as yet been produced to support this contention. However, a companion idea to this is the Hospital Service Corporation as embodied in the American Hospital Association, "Ameriplan" whereby the hospital itself would set up the corporation and utilizing its medical staff (or portion thereof it might need) offer to enroll and contract with groups or families for comprehensive care. It must be noted here that an HMO must have control of a hospital as to its cost and its practices including medical staff. At the present only Kaiser-Permanente, United Mine Workers Hospitals, some Railroad hospitals fully qualify as an HMO. Next closest come hospital-based corporations as previously mentioned or medical school-teaching hospital combination. An HMO to be effective must have such a close relationship to and with a hospital that it can always deliver at the proper cost hospital services to its enrollees and it must assure entry to the hospital to its enrollees and its staff. Therefore, while this does constitute a form of private practice in that it is non-governmental it must of necessity be subject to review and investigation particularly if set up under Federal legislation.

It must be clear by now that there is in the future of private medicine more regulations of not only fees but types of care whether rendered in the office, hospital, or nursing home. It is up to the organized physicians of this country to continue to point out that people are not all alike and that national standards of care can not be applied everywhere. In the mountain town of Provo, Utah for instance acute myocardial infarctions have been going home in 7 days while in this area the average is 13 days. The mortality rates are the same. Therefore, it must be other factors such as humidity, heat, pollution, etc. and this cannot be legislated. In 1954 faced with a closed group practice threat the physicians of San Joaquin Valley, California organized a corporation separate from but controlled by their medical society. They called it a Foundation for Medical Care whose function was to preserve the practice of medicine uncontrolled by government but indeed controlled by the physicians themselves. They set up certain guidelines of operation (1) they *would not* provide services to any insuring agent whose policies sold in their area did not meet their minimum standards (2) they *would* develop guidelines of stay in hospitals, frequency of office visits, surgery and medical care in the hospital, lab, x-ray work in or out of the hospital, drug orders and charges, etc. (3) the foundation *would* review all claims, all hospital costs, all physicians fees, and would process and pay some of them. (4) physicians' fees *would* be based on the usual and customary charges of the other physicians in the area and the maximum allowable would be known. Such a foundation functions as an educational process to all concerned; it assures physician peer review as each member rotates through as a paid reviewer, it allows the physicians to branch out into other programs such as health planning, migrant workers programs, multiphasic screening programs, and it effects an average saving as demonstrated of 11 per cent per year in the cost of health care. It is a hot item with knowledgeable members of congress. There now exist within the national organizations of foundations known as the American Association of Foundation for Medical Care 34 such foundations either fully or partially operative. This concept gives to medicine a method of preserving its independence in the face of the clamor around us.

Back to the original question—The Future of Private Medicine.

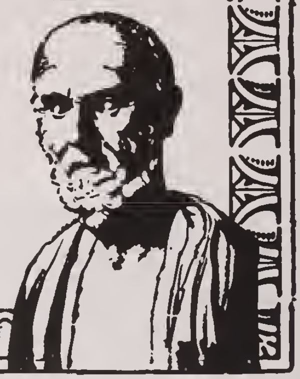
(1) Thinking members of Congress will see as has every other country in

the world which has tried it that the attempt of the central government to completely cover the people healthwise will fail and be so costly it may well bankrupt the country. It cannot be done except in a dictatorship.

(2) Medicine itself will be subjected to a more sophisticated type of review which will be broader than its present audit or tissue or utilization committees. Standardization will result as norms are established.

(3) Medicine will therefore find itself more intimately involved with such organizations as Foundations or PSRO Groups as well as Surgicenter and other localized service groups, if it remains alert, control.

(4) The future will be different yet if common sense prevails and not a utopian concept then this problem of adequate care can be solved without dislocating the present practice of medicine altho for numerous reasons already expressed there will be increasing numbers of groups either single or multi-specialty.



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Hippocrates, *Precepts*, Chapt. I

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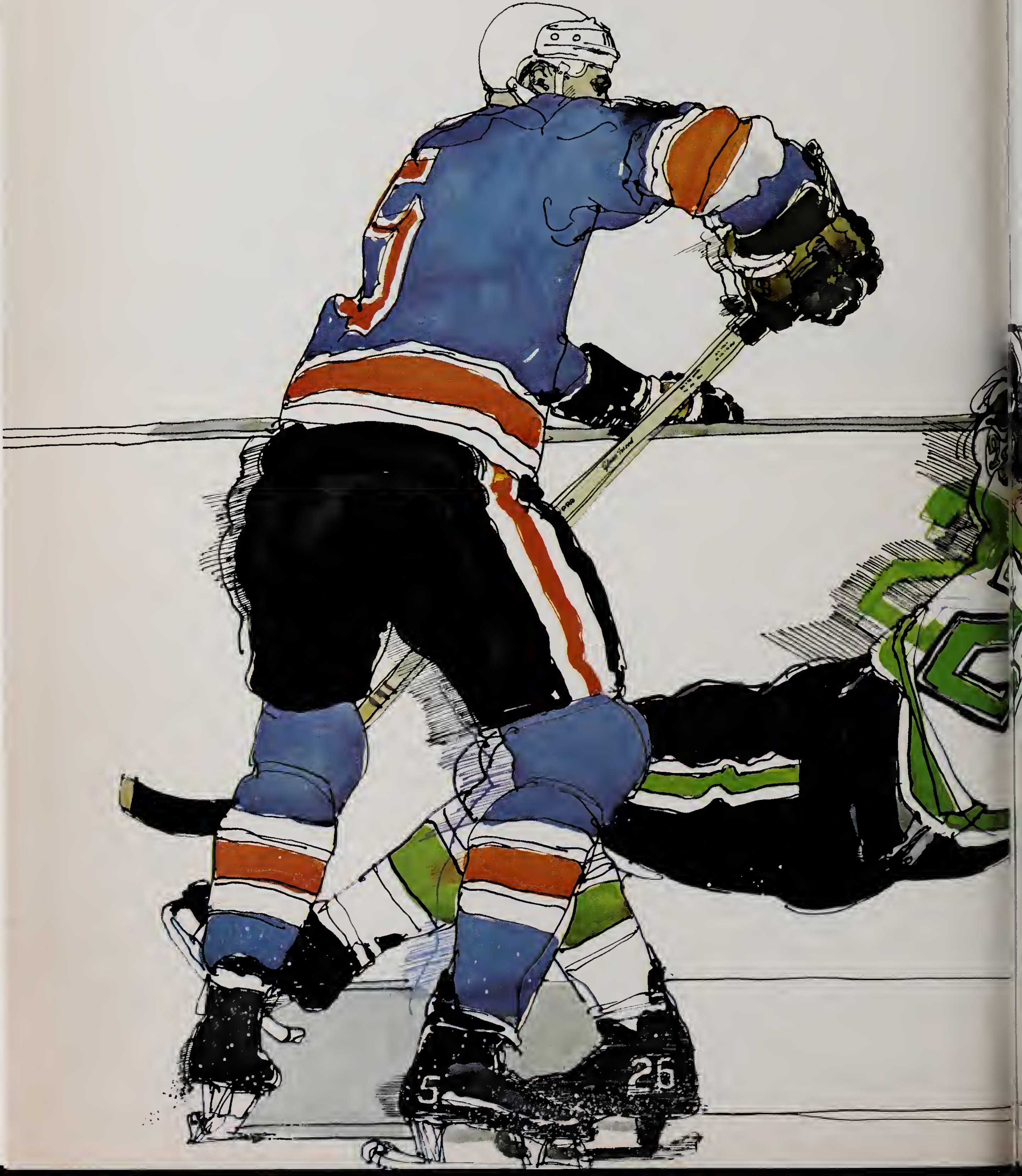

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For the skeletal muscle spasm of back sprains, Valium® (diazepam) can be a valuable adjunct. A dose of 2-10 mg, three or four times a day, goes to work to help break up the cycle of spasm/pain/spasm. The resultant relief of skeletal muscle spasm may permit greater mobilization of the affected muscles and may help the patient resume usual activities sooner than otherwise possible.

Paraspinal muscle mass frequently vulnerable to this type of trauma.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders, athetosis, stiff-man syndrome, convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence. In pregnancy, lactation or women of childbearing age, weigh potential benefit against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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A gratifying announcement about Empirin® Compound with Codeine

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It is significant in this era of increased regulation, that Empirin Compound with Codeine has been placed in a less restrictive category. You may now wish to consider Empirin with Codeine even more frequently for its predictable analgesia in acute or protracted pain of moderate to severe intensity.

Empirin Compound with Codeine No. 3 contains codeine phosphate* (32.4 mg.) gr. ½. No. 4 contains codeine phosphate* (64.8 mg.) gr. 1. *(Warning—may be habit-forming.) Each tablet also contains: aspirin gr. 3½, phenacetin gr. 2½, caffeine gr. ½.

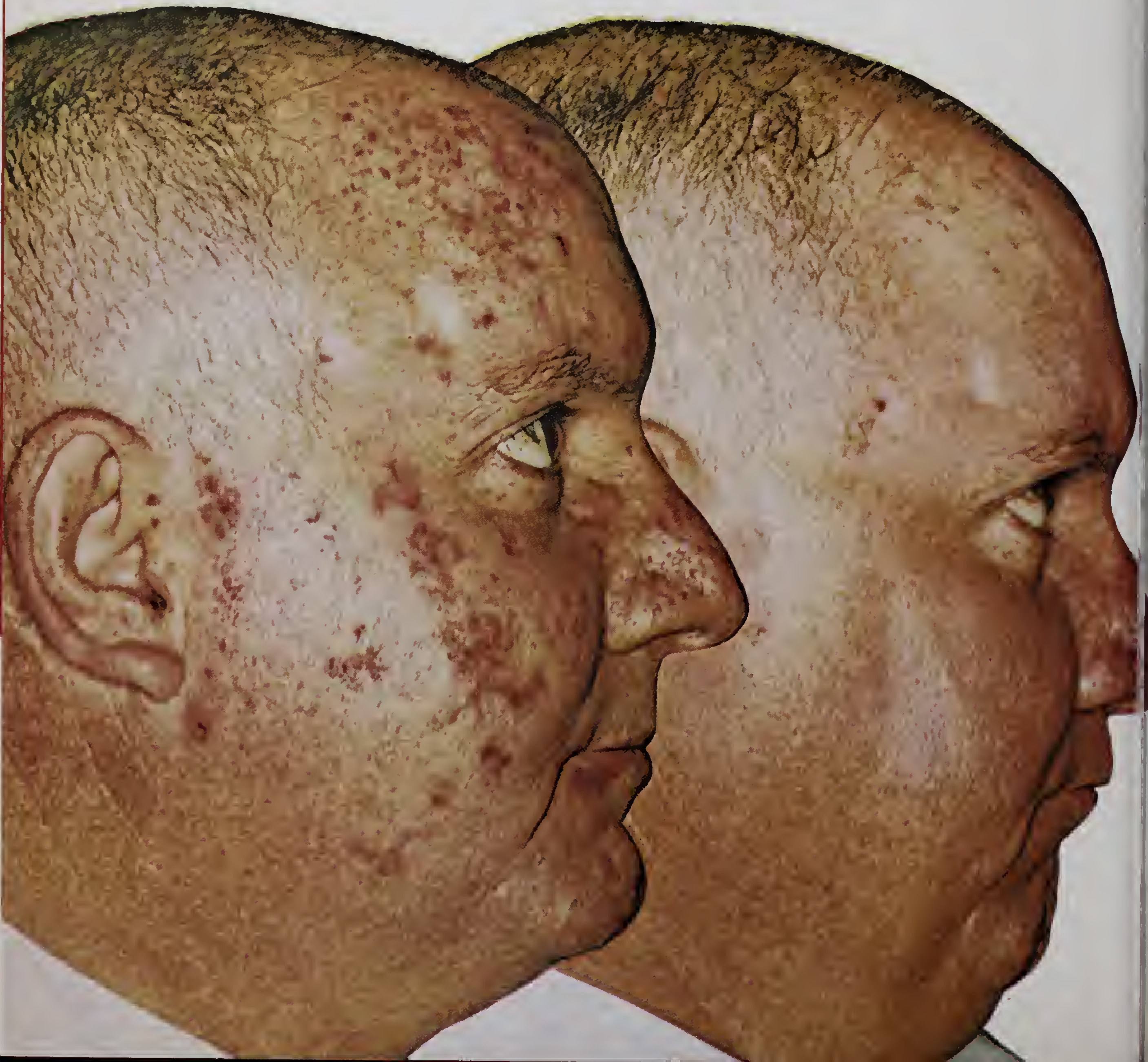
Call it what you will, it may be premalignant.

Before

3/29/67 Before therapy with 5%-FU cream. Patient P. T. shows a moderately severe solar keratotic involvement. Note residual scarring from the previous cryosurgical and electrosurgical procedures on forehead and ridge of nose adjacent to periauricular area.

After

6/12/67 Seven weeks after cessation of therapy. Reactions have subsided. Residual scarring is not seen except for that due to prior surgery. Inflammation has disappeared and face is clear of keratotic lesions.



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and Efudex® (fluorouracil) 5% cream can resolve it.

all it actinic, solar or senile keratoses, any regard it as "precancerous."[1,2]

pical fluorouracil, considered by some dermatologists to be a major lvance in the treatment of multiple solar keratoses,[3,4] offers the physi- an a relatively inexpensive alternative to cryosurgery, electrodesic- tion and cold knife surgery. Of the topical fluorouracils available, only fudex offers 2% and 5% solution and 5% cream formulations—formula- ons that have proved effective in the treatment of these mutliple lesions.

sual duration of therapy, 2 to 4 weeks.

udies showed that with the 2% and 5% Efudex preparations, the usual ration of therapy was only 2 to 4 weeks.[5] Other studies with topical orouracil revealed that when concentrations of less than 2% were ed, significant numbers of lesions recurred.[6]

reats the lesions you can't see, too.

umerous lesions, not apparent prior to 2% and 5% Efudex therapy, anifested themselves by definite reactions, while intervening skin mained relatively unaffected.[5] The early eradication of these subclini- l lesions (which may otherwise have undergone further progression) obably accounts for the reduced incidence of future solar keratoses in tients treated with topical fluorouracil—especially with 5% ncentrations.[6]

ow to identify solar keratoses.

ypically, the lesion—a flat or slightly elevated brown to red-brown apule—is dry, rough, adherent and sharply defined. Multiple lesions e the rule.

redictable therapeutic response.

he response to a typical course of Efudex therapy is usually aracteristic and predictable. After 3 or 4 days of treatment, erythema gins to appear in the area of keratoses. This is followed by a moderate intense inflammatory response, scaling and occasionally moderate nderness or pain. The height of this response generally occurs two eeks after the start of therapy and then begins to subside as treatment stopped. Within two weeks of discontinuing medication, the flammation is usually gone. Lesions that do not respond should e biopsied.

eferences: 1. Allen, A. C.: *The Skin, A Clinicopathological Treatise*, ed. 2, New York, rune & Stratton, 1967, p. 842. 2. Dillaha, C. J.; Jansen, G. T, and Honeycutt, W. M.: Treatment of Actinic Keratoses with Topical Fluorouracil," in Waisman, M. (ed.): *harmaceutical Therapeutics in Dermatology*, Springfield, Ill., Charles C Thomas, 1968, . 92. 3. Belisario, J. C.: *Cutis*, *6*:293, 1970. 4. Sams, W. M.: *Arch. Derm.*, *97*:14, 1968. . Data on file, Hoffmann-La Roche Inc., Nutley, New Jersey. 6. Williams, A. C., and lein, E.: *Cancer*, *25*:450, 1970.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Multiple actinic or solar keratoses.

Contraindications: Patients with known hypersensitivity to any of its components.

Warnings: If occlusive dressing used, may increase inflammatory reactions in adjacent normal skin. Avoid prolonged exposure to ultraviolet rays. Safe use in pregnancy not established.

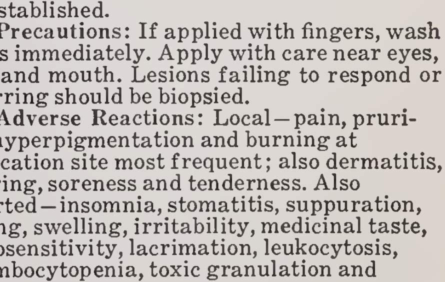
Precautions: If applied with fingers, wash hands immediately. Apply with care near eyes, nose and mouth. Lesions failing to respond or recurring should be biopsied.

Adverse Reactions: Local—pain, pruritus, hyperpigmentation and burning at application site most frequent; also dermatitis, scarring, soreness and tenderness. Also reported—insomnia, stomatitis, suppuration, scaling, swelling, irritability, medicinal taste, photosensitivity, lacrimation, leukocytosis, thrombocytopenia, toxic granulation and eosinophilia.

Dosage and Administration: Apply sufficient quantity to cover lesion twice daily with nonmetal applicator or suitable glove. Usual duration of therapy is 2 to 4 weeks.

How Supplied: Solution, 10-ml drop dispensers—containing 2% or 5% fluorouracil on a weight/weight basis, compounded with propylene glycol, tris(hydroxymethyl)aminomethane, hydroxypropyl cellulose, parabens (methyl and propyl) and disodium edetate.

Cream, 25-Gm tubes—containing 5% fluorouracil in a vanishing cream base consisting of white petrolatum, stearyl alcohol, propylene glycol, polysorbate 60 and parabens (methyl and propyl).



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The Wage-Price Freeze—Phase II

Phase II of the economic program to stabilize the domestic economy began at 12:01 a.m., November 14, 1971. The purpose of this column is to summarize and explain the most important provisions of the Phase II program in general and as it relates to physicians.

The Economic Stabilization Act of 1970, which served as the legislative basis for the 90-day freeze, is also the basis for the Phase II program. Although the Act expired on April 30, 1972, the President asked Congress to extend the Act with certain modifications until April 30, 1973; as of this writing, the Senate and the House have approved such extension and the President the same.

The principal government agencies involved in the implementation of Phase II are: the Cost of Living Council, the Pay Board and the Price Commission. The monitoring and enforcement functions of the Phase II program have been delegated to the Internal Revenue Service, which also has been assigned the duty of ruling on specific questions.

The Pay Board, which is composed of 15 members, is delegated the responsibility of formulating standards for wage and salary adjustments. A special committee will be created within the Board to deal with the problems of executive compensation.

The Price Commission consists of 7 members and is charged with performing all the functions necessary to stabilize prices and rents and with formulating the criteria for such stabilization.

Of special interest to members of the medical profession is the Committee on the Health Service Industry, which is a specialized committee consisting of representatives of the medical profession, hospitals, insurance companies, supporting industries and representatives of the general public. Its function is to advise the Pay Board and Price Commission as to special factors that tend to contribute to inflation in the health service industry. Based upon the advice of the Committee on the Health Service Industry, the Price Commission has adopted special policies with respect to the medical profession.

The general goal of the Phase II program is to stabilize prices at a level of not more than 2-1/2 per cent per year as an average general increase for the entire economy. The Committee on the Health Service Industry has determined that the current rate of inflation for health services is 12.9 per cent and its goal is to cut that rate in half. In order to accomplish this goal, prices are generally stabilized at the higher of those during the 90-day freeze period (that is, August 15–November 13, 1971) or during the period July 16–August 14, 1971. However, the special rules relating to physicians override the more general stabilization policy.

The guidelines make a distinction and provide different rules with respect to "institutional" and "non-institutional" providers of health services. Individual doctors and medical clinics fall within the non-institutional category.

A non-institutional provider of health care services may charge a price in excess of the base price only to reflect allowable costs in effect on November 14, 1971 and allowable cost increases incurred after November 14, 1971, reduced to reflect productivity gains, and only to the extent that such increased price shall not result in an increase in the non-institutional provider's profit margin (as a percentage of revenues before income taxes) over that prevailing during the base period. In addition, aggregate price increases may not exceed 2-1/2 per cent per year.

The base price is the price charged for the same service or procedure during the period August 15–November 13, 1971. The base period (used to calculate profit margin increases) is the average of any two of the non-institutional provider's last three fiscal years ending prior to August 15, 1971. At the moment, all costs are "allowable costs". However, general cost and price increases will be monitored, with the assistance of health insurers, to assure compliance with the guidelines. Further, the guidelines provide that a reduction in the quality or quantity of a service or procedure will constitute a violation of the Stabilization Program.

In general terms, a doctor (whether a sole practitioner, partnership or professional corporation) may not increase his professional or laboratory fees by more than 2-1/2 per cent. In order to justify increases up to 2-1/2 per cent, there must be cost increases to the doctor.

An additional requirement adopted by the Price Commission upon recommendation of the Committee on the Health Service Industry concerns posting. All providers of health services must maintain available for public inspection a schedule showing base prices for principal services and any change in such prices. Further, a sign must be posted in a prominent place stating the availability and location of the schedule.

Commercial real estate has been exempted from the Phase II program. Therefore, office rental increases are now permissible, except to the extent that they represent charges retroactive to the commencement of the 90-day Phase I freeze period.

In fulfilling its function as the regulator of wages and salaries, the Pay Board has adopted a general standard of limiting wage and salary increases to 5.5 per cent per year. The 5.5 per cent wage and salary standard for increases is defined very broadly. Wages and salaries include all forms of direct and indirect remuneration or inducement to employees which are reasonably subject to valuation. Nevertheless, contracts and pay practices existing on or prior to November 14, 1971 will be allowed to continue according to their terms without regard to the 5.5 per cent standard. In addition, longevity and in-grade increases pursuant to existing contracts and pay practices are also allowed without regard to the 5.5 per cent standard. Although it has been ruled that merit increases may be granted in certain circumstances, the exact application of this ruling will require further clarification.

A special committee has been established within the Pay Board to deal with the problems of executive compensation. It is expected that the pronouncements of this committee will clarify the rules as to salaries, deferred compensation and other fringe benefits.

The Phase II Regulations and policies are in a fluid state. Within a day after they were published, a host of amendments appeared. There have been, and will continue to be, numerous additional amendments. Moreover, a flood of rulings and interpretations will be issued as the Cost of Living Council, Pay Board, Price Commission, Internal Revenue Service and other units administer Phase II. Accordingly, the guidelines for price and wage increases may be altered from time to time.

JEFFREY R. REIDER, J.D.

Edited by Stefan F. Tucker, of Arent, Fox, Kintner, Plotkin & Kahn, Washington, D.C.

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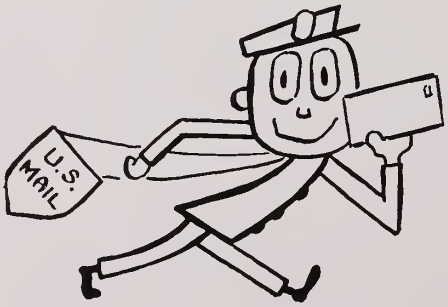

Utilization and Peer Review of the Medical Society

The Utilization Committee of the Medical Society was formed in 1965 and in 1966 met several times to establish uniform guidelines for hospital utilization committees already active or in the process of developing. At that time the Committee consisted of representatives from utilization committees of all area hospitals. Subsequently, little action was taken until 1969, when the Committee received the first cases from carriers to be reviewed. Since then it has continued to review cases at regular intervals

The purpose of the Committee is to encourage quality medical care at a reasonable cost, primarily by reviewing utilization problems. It will also arbitrate those cases in which disagreement between utilization committees of hospital and nursing homes or practising physicians and intermediaries (i.e. Medicare, Medicaid and health insurance carriers) cannot be satisfactorily resolved.

The Committee consists of 15 members of various specialties representing the attending staff of the majority of the hospitals in the city. Most of the members have served on a utilization committee of a hospital. The Committee is selected by the President of the Medical Society from the annual list of applicants for various committees.

In addition there are Peer Committees for each specialty. There are three members on each committee, with the exception of the Internal Medicine Peer Group, which consists of six physicians who are active in the Society of Internal Medicine of the District of Columbia (so with each section). The latter group has been most active in establishing communications with the carriers and rendering decisions on problem cases in their field.

The primary function of the Committee is to arbitrate cases of disputed utilization and to render decisions thereof. In addition, it shall advise and inform hospitals, nursing homes and physicians regarding methods of complying with current changes in the Medicare and Medicaid utilization laws when deemed necessary. It has also made contact with hospital utilization review committees by having a member attend one of their meetings.

The Committee receives cases for review from the carrier. The physician whose case is involved has usually been notified by the carrier that his case is to be reviewed. However, he is again notified by correspondence that the Committee will review the case. If the case involves a sub-specialty, it is referred to the respective Peer Group. After a decision has been made by the Peer Group, it is channelled to the Utilization Committee; if the decision was made by the Committee alone, it is mailed immediately to the physician in question. He is informed of the decision and asked to reply within one week, after which time the carrier will be notified. This procedure consumes more time than the operation of a single peer committee, but it has value in that it involves more of the physicians in the problems of utilization and peer review. The educational aspect of this method is beneficial.

Careful steps are taken to eliminate the name of the physician whose case is reviewed so that objective decisions can be rendered. However, when the physician wishes—or information is lacking he will be contacted. On some occasions he has met with a member of the committee. Recently this has been necessary when a physician with numerous patients in Medicare has been referred by the carrier. The reason for these reviews has been the demand by the Social Security Administration to forward cases when the Medicare income of the physician is over $25,000 per year. This type of review requires considerable time.

The Committee has also met with the carriers to establish better understanding of the type of case referred as well as to determine the role of the hospital utilization review committee with relation to Part A of Medicare. Most recently the Committee met with a representative of HEW to explain the problem of the long-term hospital Medicare case which has been approved by the hospital URC but has been retroactively denied payment by the carrier for part of this period. The Committee thinks that some progress has been made with this problem, which is a sore point to the profession as well as to the hospital. Future headway can only be made if the hospital utilization committees continue to do their chores efficiently.

Examples of cases referred include: problems of frequency of visits to nursing homes;

UTILIZATION COMMITTEE REPORT

Pictured above at a recent Utilization Committee meeting is Dr. Theodore Bedwell (second from left), Chief Medical Officer, Bureau of Health Insurance, of the Social Security Administration. He spoke on various aspects of health insurance, including the retroactive studies of Medicare.

frequent office visits over prolonged periods for conditions not requiring the same prolonged physical therapy treatment (often the Medicare patient contributes to this by benefiting from an office visit, no matter how menial the complaint is); may intermediate hospital visits for long term patients awaiting custodial care; repeated injections of medications able to be taken orally; need for hospitalization for a patient with emotional problems; and hospital visits by two physicians in the absence of an illness specifically requiring a second physician. Every consideration is given to the practicing physician in the decisions made.

The status of the federal health care program is ever changing. At present there are committees in Congress contemplating new measures for health care in the near future. It can be stated that the hospital utilization review committee now established with good performance will more than likely become a permanent fixture. The peer review mechanism is picking up momentum throughout the country as well as in Congress. The present organization of the URC of the Medical Society and its Peer Committees is such that it may be modified with little effort to meet requirements of new legislation or to be converted to a foundation.

It is apparent that the Committee will have to increase its membership in various ways to cope with the request for reviewing more cases. Only through active participation by the members of the Society can this Committee continue to function in the pursuit of quality medical care.

The Utilization Committee

Sexual Assault Program

Almost 4,000 victims of sexual assault in the District of Columbia have been assisted by the Community Health Services Administration since the program was inaugurated in September, 1965.

With sexual assault cases increasing each year in the District, the Administration's Sexual Assault Program "is of great value to both the victims and their families" according to the Metropolitan Police Department.

During 1967, the first full year of the program, more than 345 women were assisted by the program. This number has increased, and by 1971, 870 victims were assisted.

The Sexual Assault Program, initiated and directed by Dr. Charles R. Hayman, was one of the principal projects cited by the Medical Society of the District of Columbia when it presented its coveted 1971 A. H. Robins Community Service Award to Dr. Hayman. He is the first government official to receive the award, given annually to a physician for outstanding service to the community. Dr. Hayman is Associate Director for Preventive Services of the Community Health Service Administration.

The Sexual Assault Program of the Community Health Services Administration is a joint effort with the Metropolitan Police Department, in which CHSA provides medical care to women and girls and some boys who have been sexually assaulted.

Practically all assault victims have their initial medical examination at D.C. General Hospital. Some patients go to another hospital or to their own private physician. Following medical treatment, a unique nursing follow-up takes place to insure that all patients receive medical and psychiatric assistance.

Sexual assault victims are referred to the liaison public health nurse who works with the Police Department's sex squad. The nurse contacts each patient by phone, visit, or both, and if necessary, by a follow-up letter. The patient is then referred to the senior public health nurse in her district. A senior public health nurse has been selected for each of the nine nursing districts to maintain contact with the patients.

Each patient is seen or telephoned as often as indicated, in the nurse's judgment. Many patients also telephone or visit the nurse. Medical and psychiatric consultation is available to help the nurse serve the patient.

The nurse may refer patients to private physicians or public health facilities. She checks to find out if the patient has kept her appointment and what kind of treatment has been prescribed.

For several months, the nurse continues to make periodic telephone calls or visits to patients who need further counseling or referral, or whose cases are being processed further by the police, because these patients may be under great emotional strain.

The program has been well received and the nurses have observed that the patients and their families are most receptive. There appears to be a definite need for emotional support at that time. One of the major concerns of the patient and her family is the possibility of pregnancy. Venereal disease contact also is considered.

Many patients seek the nurse's aid with other problems such as housing, finances and health supervision for other members of the family. Many have had no previous contact with the Community Health Services Administration, particularly those who have moved to the District of Columbia within the year. Visits by the nurses sometimes uncover health and social problems unrelated to the sexual incident.

When negative serology and pregnancy tests are reported, the patient's anxiety decreases and so do requests for assistance.

Former Police Chief John B. Layton, in a letter to the Health Department, noted: ". . . since September 1965, when the present public health program was inaugurated to provide follow-up medical and psychiatric care for victims of sexual assault, the value of this type of assistance has been evident in several ways. The program, as presently administered, demonstrates a human interest in the welfare of victims, but more specifically, members of the sex squad have observed tangible benefits accruing from the services thus far provided. For example, sexual assault victims appear more composed and better prepared for the ordeal of a public trial. Victims are not as fearful and apprehensive regarding the possibility of pregnancy or disease after counseling by a health department representative . . . interviews and visits by the nurses have uncovered and tend to alleviate social and family problems of victims which, if allowed to develop and continue, could result in future police action. As a result of the quality and scope of the services provided by the health department for victims of sexual assault better public relation between members of the community and the police have developed as a side effect . . ."

RAYMOND L. STANDARD, M.D., M.P.H.
Director of Public Health
Associate Director, Department of Human Resources

NEW MEMBERS

Active

DAVID E. AUGUSTE, M.D. Office: Freedmen's Hospital. Born in the West Indies. Medical degree: University of the West Indies, 1962. Specialty: Internal Medicine.

SAMUEL B. AGUHOB, M.D. Office: Children's Hospital. Born in the Philippines. Medical degree: University of Santo Tomas, 1965. Specialty: Anesthesiology.

MICHAEL BRODY, M.D. Office: 3000 Connecticut Avenue. Born in Brooklyn, New York. Medical degree: New York Medical College, 1966. Specialty: Psychiatry.

RICHARD J. CASTIELLO, M.D. Office: Georgetown Hospital. Born in Washington, D.C. Medical degree: Cornell University, 1967. Specialty: Dermatology.

NAN-HUAN CHAR, M.D. Office: Children's Hospital. Born in Nanking, China. Medical degree: Kaohsiung Medical College, 1964. Specialty: Anesthesiology.

HENRY E. EDWARDS, M.D. Office: 5225 Connecticut Avenue. Born in Marianna, Ohio. Medical degree: Western Reserve University, 1962. Specialty: Psychiatry.

NORA DOCTOR JOSE, M.D. Office: Cafritz Hospital. Born in the Philippines. Medical degree: Ramon Magsaysay Medical Center, 1963. Specialty: Pediatrics.

PEDRO A. JOSE, M.D. Office: Georgetown Hospital. Born in the Philippines. Medical degree: University of Santo Tomas, 1965. Specialty: Pediatrics.

EDWIN LIEBERMAN, M.D. Office: 6451 Barnaby Street, N.W. Born in Milwaukee, Wisconsin. Medical degree: University of California, 1958. Specialty: Psychiatry.

CRESENCIANO C. LOPEZ, M.D. Office: Children's Hospital. Born in the Philippines. Medical degree: Far Eastern University, 1965. Specialty: Anesthesiology.

RODOLFO LOPEZ, M.D. Office: Hadley Memorial Hospital. Born in Batac, I.N., Philippines. Medical degree: University of Santo Tomas, 1965. Specialty: General Surgery - E.R.

JOHN JOSEPH O'CONNOR, M.D. Office: 916 19th Street, N.W. Born in Gowanda, New York. Medical degree: State University of New York at Buffalo, 1966. Specialty: Proctology.

EDUARDO SANCHEZ, M.D. Office: Cafritz Hospital. Born in Manila, Philippines. Medical degree: Cebu Institute of Medicine, 1962. Specialty: General Surgery - E.R.

STEVEN A. SCHROEDER, M.D. Office: 2150 Pennsylvania Avenue, N.W. Born in New York. Medical degree: Harvard Medical School, 1964. Specialty: Internal Medicine.

Associate

KWAN YOUNG CHOI, M.D. Office: N.W. Central Clinic. Born in Won-San, Korea. Medical degree: Chun-Nam National University, 1961. Specialty: Internal Medicine.

FACUNDO LIMA, M.D. Office: 11215 Oak Leaf Drive, Silver Spring, Maryland. Born in Cuba. Medical degree: University of Havana, 1952. Specialty: Psychiatry.

Resident-Intern

RATNAKAR L. LAWANDE, M.D. Office: D.C. General Hospital. Born in India. Medical degree: T.N. Medical College, 1966. Specialty: Otolaryngology.

Student

GEORGETOWN UNIVERSITY. Junior: Benjamin Finder.

GEORGE WASHINGTON UNIVERSITY. Junior: Jeffrey D. Harris. Seniors: Donald Clark Chapin, George M. Korengold.

Changes in Membership

Resignations

ASSOCIATE: Yu Ming Hu, M.D.

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Many people lose interest in food as they grow older. Some of them are fussy eaters—with only a few favorite foods. Others become indifferent to foods—because planning and preparing meals becomes a chore. Here Campbell's Soups can help—for these four very good reasons:

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Nourishment Campbell's Soups contain selected meats and sea foods, best garden vegetables—carefully processed to help retain their natural flavors and nutritive values.

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Recommend Campbell's Soups . . . and, of course, enjoy them yourself. Remember, *there's a soup for almost every patient and diet . . . and for every meal.*

the 3 phases of Eve

All women are not equal in their endogenous hormonal output. And, while all oral contraceptives are fundamentally effective, they exhibit **differences n their activity levels and estrogen-progestogen ratios that affect different women differently**—in both short and long-term use. Some brands may be insufficient for the woman's needs or else may exceed them.

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(*Typical clues—normal body build and breasts, feminine appearance, healthy skin and hair. Vaginal cytology slide—balanced "pink and blue.")

Some women having problems on other O.C.s might do well on Ovulen.

Ovulen has a distinctive hormonal balance that combines moderate estrogenic activity with a slight progestogen dominance. It has an excellent record of patient acceptance.

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Each white tablet contains: ethynodiol diacetate 1 mg./mestranol 0.1 mg.

References **1.** Editorial Oral Contraceptives Which Pill for Which Patient? Patient Care *3* 90-115 Feb.) 1969 and *4* 135-145 (June 15) 1970 **2.** Greenblatt, R B Progestational Agents in Clinical Practice. Med Sci *18* 37-49 (May) 1967 **3.** Kistner, R W Gynecology Principles and Practice, ed 2, Chicago, Year Book Medical Publishers, 1971 **4.** Kistner, R W The Pill: Facts and Fallacies About Today's Oral Contraceptives, New York, Delacorte Press, 1968 **5.** Nelson, J. H Clinical Evaluation of Side Effects of Current Oral Contraceptives, J Reprod Med 6:50-55 (Feb.) 1971 **6.** Orr, G W. Oral Progestational Agents Therapy and Complications, S. Dakota J Med *22* 11-17 (Jan) 1969

SEARLE

For brief summary of prescribing informati
see following page.

the Demulen phase

Many women* who secrete **more estrogen** than most do well on a pill with lower estrogen activity and an increased progestogen overbalance.

(*Typical clues—shorter, plumper, full-breasted, with glowing skin and no wrinkles. Vaginal cytology slide "pink.")

Some women with special conditions that may be aggravated by higher estrogen-activity products may do better on this ratio.

Demulen combines minimal estrogenic activity with a moderate ratio of progestogen overbalance. It is particularly well suited to the young when low-dose (activity) is preferred. Demulen offers little risk of the most potent progestogen side effects; early breakthrough bleeding is often transient.

Demulen®

Each white tablet contains ethynodiol diacetate 1 mg/ethinyl estradiol 50 mcg

Each pink tablet in Ovulen-28® and Demulen® 28 is a placebo containing no active ingredients.

Both Ovulen and Demulen are available in 21- and 28-pill schedules

the Enovid-E phase

Some women* who secrete **less estrogen** than mos
do best on a pill with a moderate estrogen overbalance.

(*Typical clues—oily complexion, acne, hirsutis
masculinity, flat chest. Vaginal cytology slide—"blue.")

Patients with estrogen deficiency may show:

premenopausal syndrome
early-cycle bleeding
scanty menses
intermittent depressio
increased appetite
steady weight gain
vaginal candidiasis

Enovid-E not only provides increased estrogeni
activity with low progestogen activity, but also contains the only progestogen that is not antiestrogenic. Therefore it offers less risk of high-dose progestogen side effects.

Enovid-E®

Each tablet contains norethynodrel 2.5 mg/mestranol 0.1 mg

Oral contraceptives are complex medications and, after reference to the prescribing information, should be prescribed with discriminating care.

for the 3 phases of Eve:

a family of O.C. products

Ovulen®

Each white tablet contains:
ethynodiol diacetate 1 mg./mestranol 0.1 mg.

Demulen®

Each white tablet contains:
ethynodiol diacetate 1 mg./ethinyl estradiol 50 mcg.

Each pink tablet in Ovulen-28® and Demulen®-28 is a placebo, containing no active ingredients.

Actions—Ovulen and Demulen act to prevent ovulation by inhibiting the output of gonadotropins from the pituitary gland. Ovulen and Demulen depress the output of both the follicle-stimulating hormone (FSH) and the luteinizing hormone (LH).

Special note—Oral contraceptives have been marketed in the United States since 1960. Reported pregnancy rates vary from product to product. The effectiveness of the sequential products appears to be somewhat lower than that of the combination products. Both types provide almost completely effective contraception.

An increased risk of thromboembolic disease associated with the use of hormonal contraceptives has now been shown in studies conducted in both Great Britain and the United States. Other risks, such as those of elevated blood pressure, liver disease and reduced tolerance to carbohydrates, have not been quantitated with precision.

Long-term administration of both natural and synthetic estrogens in subprimate animal species in multiples of the human dose increases the frequency of some animal carcinomas. These data cannot be transposed directly to man. The possible carcinogenicity due to the estrogens can be neither affirmed nor refuted at this time. Close clinical surveillance of all women taking oral contraceptives must be continued.

Indication—Ovulen and Demulen are indicated for oral contraception.

Contraindications—Patients with thrombophlebitis, thromboembolic disorders, cerebral apoplexy or a past history of these conditions, markedly impaired liver function, known or suspected carcinoma of the breast, known or suspected estrogen-dependent neoplasia and undiagnosed abnormal genital bleeding.

Warnings—The physician should be alert to the earliest manifestations of thrombotic disorders (thrombophlebitis, cerebrovascular disorders, pulmonary embolism and retinal thrombosis). Should any of these occur or be suspected the drug should be discontinued immediately.

Retrospective studies of morbidity and mortality conducted in Great Britain and studies of morbidity in the United States have shown a statistically significant association between thrombophlebitis, pulmonary embolism, and cerebral thrombosis and embolism and the use of oral contraceptives. There have been three principal studies in Britain[1-3] leading to this conclusion, and one[4] in this country. The estimate of the relative risk of thromboembolism in the study by Vessey and Doll[3] was about sevenfold; while Sartwell and associates[4] in the United States found a relative risk of 4.4, meaning that the users are several times as likely to undergo thromboembolic disease without evident cause as nonusers. The American study also indicated that the risk did not persist after discontinuation of administration and that it was not enhanced by long-continued administration. The American study was not designed to evaluate a difference between products. However, the study suggested that there might be an increased risk of thromboembolic disease in users of sequential products. This risk cannot be quantitated, and further studies to confirm this finding are desirable.

Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions medication should be withdrawn.

Since the safety of Ovulen and Demulen in pregnancy has not been demonstrated, it is recommended that for any patient who has missed two consecutive periods pregnancy should be ruled out before continuing the contraceptive regimen. If the patient has not adhered to the prescribed schedule the possibility of pregnancy should be considered at the time of the first missed period.

A small fraction of the hormonal agents in oral contraceptives has been identified in the milk of mothers receiving these drugs. The long-range effect to the nursing infant cannot be determined at this time.

Precautions—The pretreatment and periodic physical examinations should include special reference to the breasts and pelvic organs, including a Papanicolaou smear since estrogens have been known to produce tumors, some of them malignant, in five species of subprimate animals. Endocrine and possibly liver function tests may be affected by treatment with Ovulen or Demulen. Therefore, if such tests are abnormal in a patient taking Ovulen or Demulen, it is recommended that they be repeated after the drug has been withdrawn for two months. Under the influence of progestogen-estrogen preparations preexisting uterine fibromyomas may increase in size. Because these agents may cause some degree of fluid retention, conditions which might be influenced by this factor, such as epilepsy, migraine, asthma, cardiac or renal dysfunction, require careful observation. In breakthrough bleeding, and in all cases of irregular bleeding per vaginam, nonfunctional causes should be borne in mind. In undiagnosed bleeding per vaginam adequate diagnostic measures are indicated. Patients with a history of psychic depression should be carefully observed and the drug discontinued if the depression recurs to a serious degree. Any possible influence of prolonged Ovulen or Demulen therapy on pituitary, ovarian, adrenal, hepatic or uterine function awaits further study. A decrease in glucose tolerance has been observed in a significant percentage of patients on oral contraceptives. The mechanism of this decrease is obscure. For this reason, diabetic patients should be carefully observed while receiving Ovulen or Demulen therapy. The age of the patient constitutes no absolute limiting factor, although treatment with Ovulen or Demulen may mask the onset of the climacteric. The pathologist should be advised of Ovulen or Demulen therapy when relevant specimens are submitted. Susceptible women may experience an increase in blood pressure following administration of contraceptive steroids.

Adverse reactions observed in patients receiving oral contraceptives—A statistically significant association has been demonstrated between use of oral contraceptives and the following serious adverse reactions: thrombophlebitis, pulmonary embolism and cerebral thrombosis.

Although available evidence is suggestive of an association, such a relationship has been neither confirmed nor refuted for the following serious adverse reactions: neuro-ocular lesions, e.g., retinal thrombosis and optic neuritis.

The following adverse reactions are known to occur in patients receiving oral contraceptives: nausea, vomiting, gastrointestinal symptoms (such as abdominal cramps and bloating), breakthrough bleeding, spotting, change in menstrual flow, amenorrhea during and after treatment, edema, chloasma or melasma, breast changes (tenderness, enlargement and secretion), change in weight (increase or decrease), changes in cervical erosion and cervical secretions, suppression of lactation when given immediately post partum, cholestatic jaundice, migraine, rash (allergic), rise in blood pressure in susceptible individuals and mental depression.

Although the following adverse reactions have been reported in users of oral contraceptives, an association has been neither confirmed nor refuted: anovulation post treatment, premenstrual-like syndrome, changes in libido, changes in appetite, cystitis-like syndrome, headache, nervousness, dizziness, fatigue, backache, hirsutism, loss of scalp hair, erythema multiforme, erythema nodosum, hemorrhagic eruption and itching.

The following laboratory results may be altered by the use of oral contraceptives: hepatic function: increased sulfobromophthalein retention and other tests; coagulation tests: increase in prothrombin, Factors VII, VIII, IX and X; thyroid function: increase in PBI and butanol extractable protein bound iodine, and decrease in T^3 uptake values; metyrapone test and pregnanediol determination.

References: 1. Royal College of General Practitioners: Oral Contraception and Thrombo-Embolic Disease, J. Coll. Gen. Pract. *13*:267-279 (May) 1967. **2.** Inman, W. H. W., and Vessey, M. P.: Investigation of Deaths from Pulmonary, Coronary, and Cerebral Thrombosis and Embolism in Women of Child-Bearing Age, Brit. Med. J. *2*:193-199 (April 27) 1968. **3.** Vessey, M. P., and Doll, R.: Investigation of Relation Between Use of Oral Contraceptives and Thromboembolic Disease. A Further Report, Brit. Med. J. *2*:651-657 (June 14) 1969. **4.** Sartwell, P. E.; Masi, A. T.; Arthes, F. G.; Greene, G. R., and Smith, H. E.: Thromboembolism and Oral Contraceptives: An Epidemiologic Case-Control Study, Amer. J. Epidem. *90*:365-380 (Nov.) 1969.

SEARLE Products of SEARLE & CO.
San Juan, Puerto Rico 00936

Enovid-E®

norethynodrel 2.5 mg./mestranol 0.1 mg.

Actions—Enovid-E acts to prevent ovulation by inhibiting the output of gonadotropins from the pituitary gland. Enovid-E depresses the output of both the follicle-stimulating hormone (FSH) and the luteinizing hormone (LH).

Indication—Enovid-E is indicated for oral contraception.

The *Special Note, Contraindications, Warnings, Precautions* and *Adverse Reactions* listed above for Ovulen and Demulen are applicable to Enovid-E and should be observed when prescribing Enovid-E.

SEARLE **Enovid-E®**
brand of norethynodrel with mestranol

Product of G. D. Searle & Co.
P.O. Box 5110, Chicago, Illinois 60680
Where "The Pill" Began

Old winner, new bottle.

DBI® phenformin HCl
tablets of 25 mg.
DBI-TD® phenformin HCl
capsules of 50 and 100 mg.

Indications: Stable adult diabetes mellitus; sulfonylurea failures, primary and secondary; adjunct to insulin therapy of unstable diabetes mellitus. *Contraindications:* Diabetes mellitus that can be regulated by diet alone; juvenile diabetes mellitus that is uncomplicated and well regulated on insulin; acute complications of diabetes mellitus (metabolic acidosis, coma, infection, gangrene); during or immediately after surgery where insulin is indispensable; severe hepatic disease; renal disease with uremia; cardiovascular collapse (shock); after disease states associated with hypoglycemia. *Warnings:* Use during pregnancy is to be avoided. *Precautions:* 1. *Starvation Ketosis:* This must be differentiated from "insulin lack" ketosis and is characterized by ketonuria which, in spite of relatively normal blood and urine sugar, may result from excessive phenformin therapy, excessive insulin reduction, or insufficient carbohydrate intake. Adjust insulin dosage, lower phenformin dosage, or supply carbohydrates to alleviate this state. **Do not give insulin without first checking blood and urine sugar.**
2. *Lactic Acidosis:* This drug is not recommended in the presence of azotemia or in any clinical situation that predisposes to sustained hypotension that could lead to lactic acidosis. To differentiate lactic acidosis from ketoacidosis, periodic determinations of ketones in the blood and urine should be made in diabetics previously stabilized on phenformin, or phenformin and insulin, who have become unstable. If electrolyte imbalance is suspected, periodic determinations should also be made of electrolytes, pH, and the lactate-pyruvate ratio. The drug should be withdrawn and insulin, when required, and other corrective measures instituted immediately upon the appearance of any metabolic acidosis.
3. *Hypoglycemia:* Although hypoglycemic reactions are rare when phenformin is used alone, every precaution should be observed during the dosage adjustment period particularly when insulin or a sulfonylurea has been given in combination with phenformin.
Adverse Reactions: Principally gastrointestinal; unpleasant metallic taste, continuing to anorexia, nausea and, less frequently, vomiting and diarrhea. Reduce dosage at first sign of these symptoms. In case of vomiting, the drug should be immediately withdrawn. Although rare, urticaria has been reported, as have gastrointestinal symptoms such as anorexia, nausea and vomiting following excessive alcohol intake.
(B)98-146-103-C

DBI-TD®
phenformin HCl
Geigy

For complete details, including dosage, please see full prescribing information.

GEIGY Pharmaceuticals
Division of CIBA-GEIGY Corporation
Ardsley, New York 10502
Distributors

DBI 8123-9

This winter
the islets of

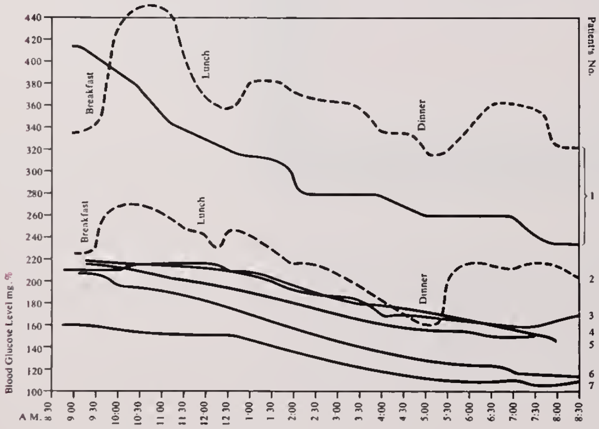
Let's say you've decided that diet, weight loss, and insulin won't work in your adult-onset, nonketotic diabetic. You're considering DBI-TD or a sulfonylurea. Which?

For a new patient, it's your choice. Both lower blood sugar. DBI-TD does it without stimulating insulin from the pancreas. Actually, it may decrease insulin oversecretion.

And if you start with DBI-TD... there's once-a-day dosage for many patients, and a convenient range of dosage forms: 100-mg. and 50-mg. capsules, and 25-mg. DBI tablets.

For sulfonylurea failures (primary or secondary): Since DBI-TD doesn't work on the islets, you may want to add it to sulfonylureas. Or replace them entirely.

Continuous chemical monitoring of blood glucose: DBI-TD (timed-disintegration capsules)



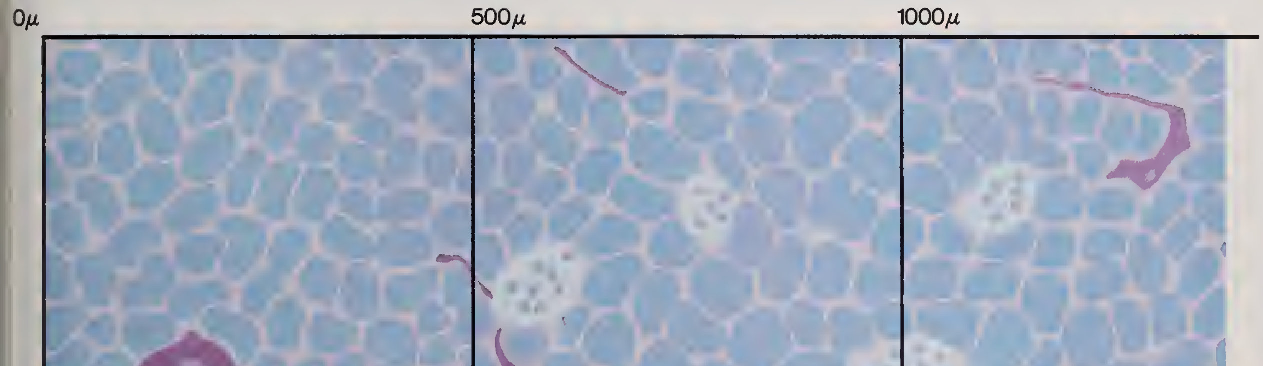
A single dose of the controlled-release form produced a hypoglycemic effect apparent in 15 minutes and lasting for at least 12 hours. Patients 2, 5, 6, 7—50 mg. Patients 1, 3, 4—100 mg.

Blood glucose continuously measured in seven ketoacidosis-resistant diabetics who had not received oral hypoglycemic agents or insulin for 48 hours; some were fasting, others were fed. *Weller, C. and Linder, M.: Metabolism 10*:669, 1961.

Before using DBI-TD or any oral hypoglycemic, please refer to the prescribing information concerning warnings, contraindications, precautions and adverse reactions. See summary on preceding page of this ad.

DBI-TD® Geigy
phenformin HCl

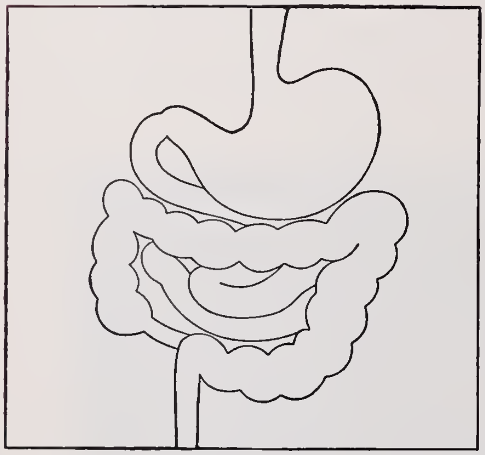
why go to Langerhans?


0µ
500µ
1000µ
Islets of Langerhans
Interlobular Tissue

in

“Gasspastic”

conditions



The GI tract in spasm is commonly a “gas trap.”

Sidonna® is formulated to release entrapped gas, as well as to provide antispasmodic/sedative effects.

In addition to the traditional combination of belladonna alkaloids and butabarbital (warning: may be habit forming.), Sidonna contains simethicone—a non-systemic defoaming agent that “lyses” gas bubbles on contact.

Sidonna has the ability to relieve GI spasm, pain **and gas** in the irritable bowel syndrome, spastic colon, pylorospasm, gastroenteritis, gastritis, nausea, nervous indigestion, or gastric and duodenal ulcer.

Sidonna can calm GI spasm...control anxiety...and release entrapped GI gas from the system.

Sidonna can do more for your “gasspastic” patient. Try him on 1 or 2 tablets before meals and at bedtime.

Sidonna®

Each scored tablet contains: Specially activated simethicone 25 mg.; hyoscyamine sulfate 0.1037 mg., atropine sulfate 0.0194 mg., hyoscine hydrobromide 0.0065 mg. (equivalent to belladonna alkaloids [as bases] 0.1049 mg.) and butabarbital sodium N.F. 16 mg. (Warning: May be habit forming.)

can do more

Contraindications: Anticholinergics should not be used in patients with glaucoma, known prostatic hypertrophy, or pyloric obstruction. Urinary retention may indicate the presence of prostatic hypertrophy. If it occurs, the dose should be reduced or the drug withdrawn. Also contraindicated in patients with known hypersensitivity to one of the components.
Side Effects: Dryness of the mouth, blurred vision, dysuria, skin rash, constipation or drowsiness may occur.

Reed & Carnrick/ Kenilworth, New Jersey 07033

"For generations my family has insisted on Donnagel®-PG," says active young matron Mrs. T. Farnsworth Lipp (of the Upper Lipps), shown here with her charming son. "All the benefits of paregoric—without the unpleasant taste, don't you know? And Junior thinks Donnagel-PG tastes so much like bananas that I never worry about a slip between spoon and Lipp."

A Matter of Taste

With or without a silver spoon, a most tasteful solution in treating acute, non-specific arrheas: all the benefits of paregoric, without the unpleasant taste. Donnagel®-PG treats companying cramping, tenesmus, and nausea as well as the diarrhea itself. Instead of pleasant-tasting paregoric, it contains the therapeutic equivalent, powdered opium, promote the production of formed stools and lessen the urge. And it provides the mulcent-detoxicant effects of kaolin and pectin, plus the antispasmodic benefits of lladonna alkaloids. And a good banana flavor to baby any taste.

Donnagel-PG
8 FL.OZ.

Donnagel®-PG
Donnagel with paregoric equivalent
Ⓥ Available on oral prescription or without prescription under limited circumstances as modified by applicable state law.

Each 30 cc. contains: Kaolin, 6.0 g.; Pectin, 142.8 mg.; Hyoscyamine sulfate, 0.1037 mg.; ropine sulfate, 0.0194 mg.; Hyoscine hydrobromide, 0.0065 mg.; Powdered opium, USP, 24.0 mg. quivalent to paregoric 6 ml.) (Warning: may be habit forming); Sodium benzoate (preservative), 0 mg.; Alcohol, 5%. A.H. Robins Company, Richmond, Virginia 23220

A·H·ROBINS

coughs are back...
4 FL OZ
Robitussin
Robitussin-DM
Robitussin
4 FL OZ
Robitussin A-C
Cough Calmers

clear the tract!

The coughing season is here again. Time to rely on the four Robitussins and Cough Calmers to help clear the lower respiratory tract. All contain glyceryl guaiacolate, the efficient expectorant that works systemically to help increase the output of lower respiratory tract fluid. The enhanced flow of less viscid secretions soothes the tracheobronchial mucosa, promotes ciliary action, and makes thick, inspissated mucus less viscid and easier to raise. Available on your prescription or recommendation.

For coughs of colds and "flu"

Robitussin®

Each 5 cc. contains:

Glyceryl guaiacolate	100.0 mg.
Alcohol, 3.5%	

For unproductive allergic coughs

Robitussin A-C®

Each 5 cc. contains:

Glyceryl guaiacolate	100.0 mg.
Pheniramine maleate	7.5 mg.
Codeine phosphate	10.0 mg.
(warning: may be habit forming)	
Alcohol, 3.5%	

Non-narcotic for 6-8 hr. cough control

Robitussin-DM®

Each 5 cc. contains:

Glyceryl guaiacolate	100.0 mg.
Dextromethorphan hydrobromide	15.0 mg.
Alcohol, 1.4%	

Clears sinuses and nasal stuffiness as it relieves cough

Robitussin-PE®

Each 5 cc. contains:

Glyceryl guaiacolate	100.0 mg.
Phenylephrine hydrochloride	10.0 mg.
Alcohol, 1.4%	

Robitussin-DM in solid form for "coughs on the go"

Cough Calmers®

Each Cough Calmer contains:

Glyceryl guaiacolate	50.0 mg.
Dextromethorphan hydrobromide	7.5 mg.

with the ROBITUSSIN® LINE

lect the Robitussin® "Clear-Tract" Formulation That Treats Your Patient's Individual Coughing Needs:

All 5 Robitussins have an EXPECTORANT-DEMULCENT action. Keep this handy chart as a guide in selecting the formula that provides the *extra* benefits you want for your patient.

bitussin® extra efit chart	Cough Suppressant	Antihistamine	Long-Acting (6-8 hours)	Nasal, Sinus Decongestant	Non-Narcotic
OBITUSSIN®					●
OBITUSSIN A-C®	●	●			
OBITUSSIN-DM®	●		●		●
OBITUSSIN-PE®				●	●
OUGH CALMERS®	■		■		■

A. H. Robins Company,
Richmond, Virginia 23220
A·H·ROBINS

℞
Meltrol-50
Caps # 100
Sig

INTRODUCING
Meltrol-50™
the **new** USV brand of
phenformin HCl

Meltrol-50 (phenformin HCl)
50 mg. timed-disintegration capsules
also Meltrol-100™
(100 mg. timed-disintegration capsules)
Meltrol-25™ (25 mg. tablets)

USV PHARMACEUTICAL CORP., Tuckahoe, N.Y. 10707

FROM
THE NEW
USV

d.c. MENTAL HEALTH ADMINISTRATION

Area C Community Mental Health Center and the Development of Outreach Teams

". . . . .Community psychiatry referring to those programs in which psychiatric leadership prevails and "community mental health" being a more generic term, without reference to professional or disciplinary leadership. . . Both community psychiatry and community mental health may be defined as involving the application of relevant behavioral and social sciences (including social psychiatry) in order to investigate and to meet the mental health needs of a functionally or geographically delineated community."[1] Melvin Sabshin.

Sabshin in defining community psychiatry and community mental health has described elements of a developmental process which has occurred at Area C Community Mental Health Center over a period of years. The Center-based program gradually has located increasing amounts of its staff in neighborhood locations. Interest of various staff members and in specific instances at the request of community residents both mental health teams and individual mental health workers have moved into community clinics and apartments to serve specific limited geographic locations in catchment Area C. Area C includes portions of the four major sections of Washington radiating out from the Capitol: e.g. all of Southwest, most of Northeast, a small portion of inner city Northwest and that portion of Southeast located west of the Anacostia River. In early 1968 when Center staff became increasingly interested in finding neighborhood bases of operation, a concern for the area in which outreach teams would operate was one of a series of administrative problems. This difficulty was lessened when the Mayor designated nine city service areas for the development of numerous decentralized consumer services. Team development has since related to these city divisions.

Currently four outreach teams are operating in Area C serving different sections of the catchment area. Three are run with a combination of full and part-time staff; the fourth has been part-time since 1968 but anticipates having full time staff this year. Teams are composed of staff from various professional disciplines and representing the range of service programs operated at the Center. Thus all teams have the part-time services of a psychiatrist, as well as services of social workers, nurses, social work associates and representatives and secretary on full time teams. Teams also have available part-time services of psychologists, occupational therapists and rehabilitation workers.

The development of teams and individual mental health workers from staff who have traditionally operated in more institutionalized settings has produced a series of challenges to the Center and to the staffs moving into the community. Among these have been the need of all staff for orientation to and continuing education about the community they serve, the responsibility for administrative liaison with the Center, the diversity of their daily roles as community mental health workers in comparison with the more static role of Center-based mental health disciplines and the need to develop career ladder opportunities for the new community workers.

The types of requests brought to community workers has been partially described by Lucy Cohen, Ph.D. in 1969.[2] "Mental Health workers have received various types of referrals from the neighborhood center staff, community agency representatives, and the citizenry. Referrals for screening purposes, parent-child relationship problems, school behavior and acting out problems, among others, have all been part of the gamut of services requested." A description of one of the teams specifically gives a closer look at their services to a particular community. Each team has a distinctive character and this contributes to variety in operation which hopefully leads to best serving the needs of the neighborhood in which each is located.

The Southwest Team, an outreach team developed from Area C Community Mental Health Center; is based in an apartment in a public housing project. It is responsible for the delivery of mental health services to Southwest Washington, an area of approximately 16,000 people whose population is housed in public housing, individual homes and townhouses as well as high income high rise apartments. The team was organized in response to a request of area community leaders based in the Southwest Community House. Representatives from the community began interviewing Center staff on December 30, 1969 and selected the eventual team members. Temporary space was soon provided for the team by the Southwest Community House. Permanent space was made available on July 1, 1970 in an apartment in a public housing project with the assistance of pressure from community members.

This team and each of the others is composed of professionals from various disciplines and programs at the Center. Full time staff include a nurse, a social worker, 2 social work associates and 5 social work representatives, an occupational therapist and a secretary. Part-time team members include a psychiatrist, a psychologist and vocational rehabilitation counsellor. Staff members will serve anyone who lives in the Southwest and requests service. No age or financial limits or any other requirements exist. Referrals for residents of the area are likewise accepted from any agency or individual in the area such as private physicians.

Help offered may include individual therapy, group therapy, groups in the public schools, activity groups, family and social network oriented services, therapy within the homes of community residents, help to local neighborhood interest groups who meet concerning local family and community life. Services are offered to all family members and include such special areas as problems of aging, adolescence and alcoholism. Staff serve as consultants to day-care services for pre-schoolers, and other agency professionals such as school counsellors. Several of these agencies may apply directly to a given client and his family. For example service to an adolescent referred to the team includes offering help in clarifying and resolving the realities of his current living situation. The team member making contact with the youth may discover needs for a variety of requirements in his particular situation. In addition to offering individual and/or group therapy as necessary, discussion may reveal a need for such activities as special educational needs, job opportunities, training through D.V.R., and recreational planning. The local branch of ACT, U.S.E.S. or the school may be contacted by the team member to assist. The team member may continue to see the youth and usually his family while he may be encouraged to contact other local services such as Southwest House. The team member may serve as advocate for the youth and his family in such activities as identifying, contacting, and locating more appropriate housing or needed legal aid. If requested the team member may accompany a client in their efforts to obtain such services.

Team members also participate in community functions such as tenant's councils, the Southwest Assembly and Southwest Health Clinic. In general they actively participate in community efforts such as community meetings for improvement in overall community life.

For physicians and other persons wishing to contact the outreach teams they may be reached at the following locations.

Telephone	*Location*
544-0213	Arthur Capper Mental Health Workers 1101 7th Street, S.E. W. Augustus Reid, M.D. Supervisory Psychiatrist Toni Francis, Coordinator
399-4824	Parkside Mental Health Workers 701 Kenilworth Terrace, N.E. W. Augustus Reid, M.D. Supervisory Psychiatrist Shirley Ford, Coordinator
544-1715	Southwest Mental Health Workers 1200 Delaware Avenue, S.W. Vincent Kelly, M.D. Supervisory Psychiatrist Ruth Ricks, Coordinator
382-1065	Northwest One Mental Health Workers 941 North Capitol Street, N.E. Luke Grande, M.D. Supervisory Psychiatrist Rachel Geiger, Coordinator

December 3, 1971

ESSEX C. NOEL, III, M.D.
Director, D.C. Mental Health Administration

References

1. ROBERT, LEIGH, GREENFIELD, NORMAN, MILLER, MILTON, eds., *Comprehensive Mental Health, the Challenge of Evaluation*, University of Wisconsin Press, Madison: 1968 pp. 20.
2. Cohen, Lucy, Ph.D., "The Mental Health Worker in the Neighborhood Health Centers, Area C: Some Preliminary Considerations," Department of Public Health, Washington, 1969.

book REVIEWS

Stereoscopic Atlas of Macula Diseases—A Funduscopic and Angiographic Presentation. J. DONALD M. GASS, M.D. Price, $48.50. Pp. 323, with 498 illustrations, 105 stereoscopic views, View Master Reels and compact viewer. St. Louis: C. V. Mosby Co., 1970.

By selecting stereoscopic kodachrome slides, as well as stereoscopic fluorescein angiographic transparencies to supplement many black-and-white photographs, this atlas achieves an in vivo quality of presentation lacking in even the best of conventional color print texts. This is the significant virtue of this publication.

The classification of maculopathies is practical, according to associated intraocular structures involved: choroidal diseases productive of localized detachment, retinal pigment epithelium disease, non-exudative heredodegenerative diseases of the choroid, diseases of the pigment epithelium, retinal vascular diseases, retinal inflammatory diseases, trauma, neoplastic disease of the retina, non-exudative retinal degenerative disease, vitreous abnormalities, congenital pits of the optic nerve.

The range of material will extend the clinical experience of most ophthalmologists beyond their usual practice encounters. This is the second significant virtue of the text. It is likely to be the practitioner of numerous years experience or the ophthalmologist who is the product of postresidency fellowship training who has been privileged to observe all the varieties of maculopathies photographed so carefully here, through their varying stages of complete evolution.

The supplementary text in this atlas, as in all others, is necessarily of a summary nature. This may account for some unqualified generalizations and poorly substantiated opinions about currently popular modes of therapy, such as photocoagulation, based on single case experiences reported, where final judgement might be withheld until more extensive data are accumulated. The emphasis upon pathogenesis, pathology and therapy is uneven for different subjects, but the reader is supplied with reference sources. As he has done in his other works, Dr. Gass provides helpful diagrammatic figures to relate histologic with ophthalmoscopic features and angiographic appearance pathogenic interpretation.

Careful study of the stereoscopic slides will increase the ophthalmologist's diagnostic acumen.

WILLIAM S. GILBERT, M.D.

The Medical Offenders. HOWARD & MARTHA LEWIS. Price, $7.95. Pp. 377. New York: Simon & Schuster, 1970.

The success and failure of any book must be measured by the achievement of its objectives. The title "The Medical Offenders" would suggest that the authors' intent was to catalogue the varied frailties of individual medical practitioners. This they succeed in doing, and in the process they regurgitate forth chapter after chapter of the physician as fee gouger, sex offender, narcotic addict, perjurer, quack, etc., etc. (it is painful to continue).

The authors, Howard and Martha Lewis, present colorful dialogue, complete with direct quotes of purported conversations between patient and physician, physician and physician, etc. Many of these alleged conversations strain credibility, but since this book is a work of non-fiction one can only admire their reporting efforts, in preserving such monumental prose as the pleading patient's "Don't touch me" inveighing against the addicted physician's advances.

Is this compendium of physician evil the book that the Lewis' wanted to write? Or, are they carried away by their voluminous files on physician error and mischief which they spew forth in an appendix which relies heavily on reference from the 1950's. In their text they seldom bother to indicate chronology so that references to a medical "expose" in the Washington Star implies current happening rather than indicating that the article was written in 1959.

The problem with this book is that the meaningful discussion of medical relicensure is blurred by the authors' tendency to "pour it on" just as they claim medical overtreaters do. Their disclaimer that they recognize physician misdeed is a problem with a small number of physicians, cannot correct the distorted focus of this work. Instead of being primarily an objective analysis of the problems of physician recertification and what is being done to upgrade the profession such as medical education, self-assessment, and peer review—to name a few the authors prefer to magnify the human failings of a small number of individuals.

This is not a well-balanced, even-handed, fair book about physicians. This is a book about physicians' warts, and old rehashed warts at that!!

RAYMOND SCALETTAR, M.D.

The **Arthritis Handbook: A Patient's Manual on Arthritis, Rheumatism and Gout.** DARRELL C. CRAIN, M.D., F.A.C.P. Second Edition, revised. Price, $6.50 Pp. 220 with illustrations. New York: Exposition Press, 1971.

Dr. Crain has produced a second edition of his Patient's Manual on Arthritis, Rheumatism and Gout. The first edition appearing in 1959 was a paperback and served a useful function in making available an easily read and understood treatise for the patient. The second edition, with a change in title to make its appeal directly on being read, is a welcome contribution to lay-directed literature. Chapters I to VII give brief, succinct descriptions of the various forms of joint disease except gout so that the afflicted reader can get a good notion as to which type of joint disease is troubling him. In these descriptions, the reviewer felt that at times there was more use than desirable of technical terms, but substitute lay terminology is difficult to find. Chapters IX and X discuss what can be done and medications available.

Part II consisting of Chapters XIII to XX discuss gout. In these chapters, as in the chapters on the other inflammatory arthritides, the author properly stresses the systemic nature of the disease despite its often localized manifestation. This is a useful, desirable concept for the lay reader to get.

The third section, consisting of Appendices A, B and C on Diets, Exercises and Appliances, respectively are very welcome additions to patients' knowledge. Good nutrition is given a proper place and the answers to, "Doctor, what shall I eat?" are given. The appendices on Exercises and Appliances are well illustrated and explicit.

All in all, the volume is a timely and useful addendum to what physicians can offer the patient as adjuncts to his therapy. It is regrettable that it is produced in hard cover and not in paper cover. Paper cover would surely, in the opinion of the reviewer, make for much wider distribution of this volume. Dr. Crain is to be thanked and commended for his contribution.

MYER H. STOLAR, M.D.

OBITUARIES

Thomas G. Edison, M.D.
(1920-1971)

Dr. Thomas G. Edison, a Washington area surgeon, died November 28, 1971. He lived at 9629 Old Spring Road, Kensington, Maryland.

Born in St. Louis, Missouri on October 29, 1920, he received both his bachelor of arts and his medical degree from Washington University. He interned at the U.S. Naval Hospital in Chelsea, Massachusetts and Gallinger Hospital. He was a resident at Gallinger Hospital, George Washington University Hospital, and Childrens Hospital. He served in the medical corps of the Naval Reserve between 1945 and 1948.

Dr. Edison was a member of the Certified American Board of Surgery, Fellow American College of Surgeons, Southern Medical Association, and the Washington Academy of Surgery.

He is survived by his wife, the former Katherine Fisher; two children, Noel Cochran and Holly, all of the home address, and his father, George Edison of St. Louis.

Of GENERAL INTEREST

A Job Well Done

Henry D. Ecker, M.D., Outgoing Chairman of the Executive Board (left) is shown here presenting the President's gavel to Marvin C. Korengold, M.D. for his outstanding job as President of the Society during the year 1971. The presentation was made at the Annual Business Meeting held in November.

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unwelcome bedfellow for any patient– including those with arthritis, diabetes or PVD

- ☐ Prevents painful night leg cramps
- ☐ Permits restful sleep
- ☐ Provides simple convenient dosage — usually just one tablet at bedtime

Prescribing Information — Composition: Each white, beveled, compressed tablet contains: Quinine sulfate, 260 mg., Aminophylline, 195 mg. **Indications:** For the prevention and treatment of nocturnal and recumbency leg muscle cramps, including those associated with arthritis, diabetes, varicose veins, thrombophlebitis, arteriosclerosis and static foot deformities. **Contraindications:** Quinamm is contraindicated in pregnancy because of its quinine content. **Precautions/Adverse Reactions:** Aminophylline may produce intestinal cramps in some instances, and quinine may produce symptoms of cinchonism, such as tinnitus, dizziness, and gastrointestinal disturbance. Discontinue use if ringing in the ears, deafness, skin rash, or visual disturbances occur. **Dosage:** One tablet upon retiring. Where necessary, dosage may be increased to one tablet following the evening meal and one tablet upon retiring. **Supplied:** Bottles of 100 and 500 tablets.

Merrell

MERRELL-NATIONAL LABORATORIES
Division of Richardson-Merrell Inc.
Cincinnati, Ohio 45215

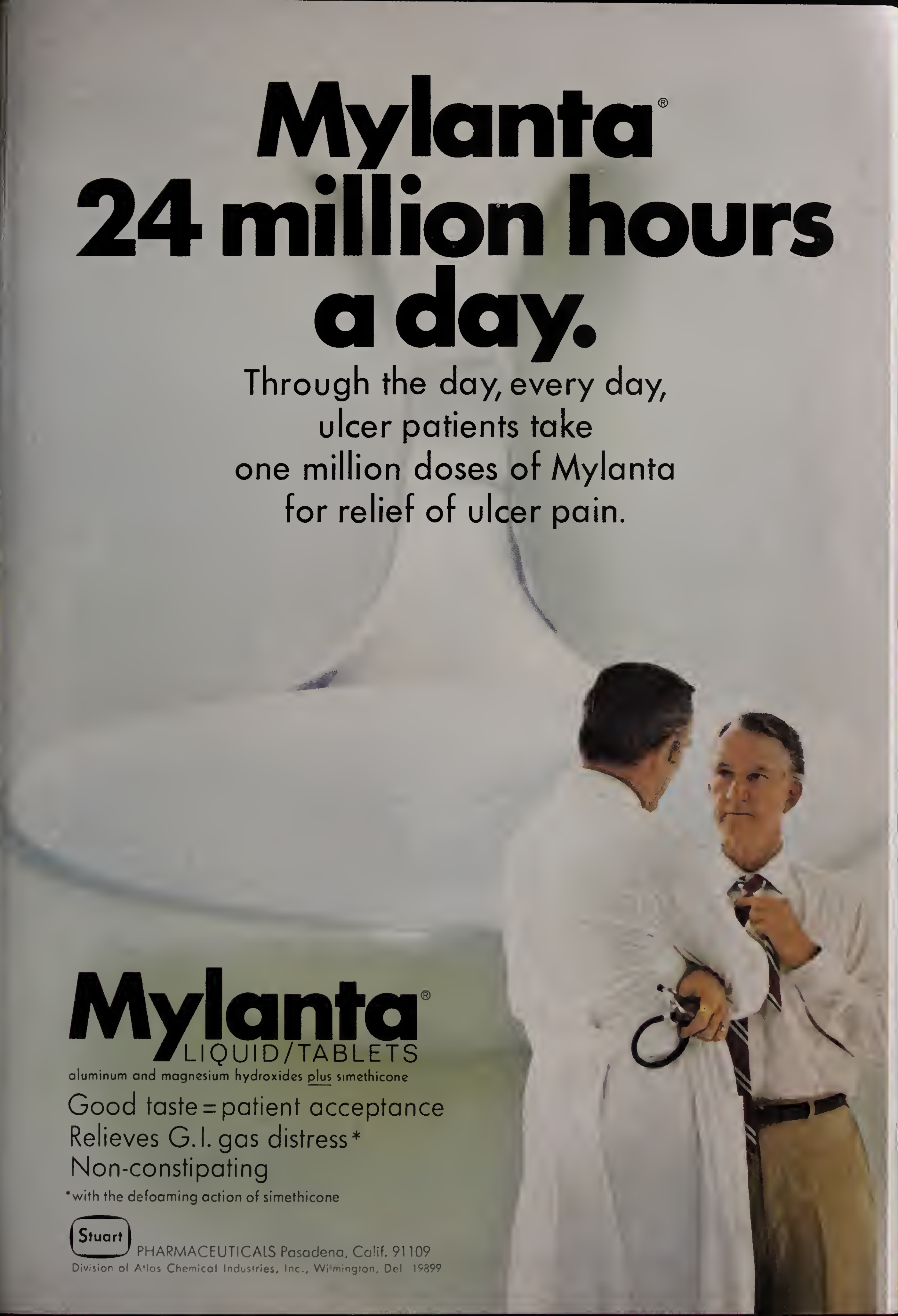
1-3508(3050)

Trademark: Quinamm

Quinamm

(quinine sulfate 260 mg., aminophylline 195 mg.)

Specific therapy for night leg cramps.



Mylanta®
24 million hours a day.

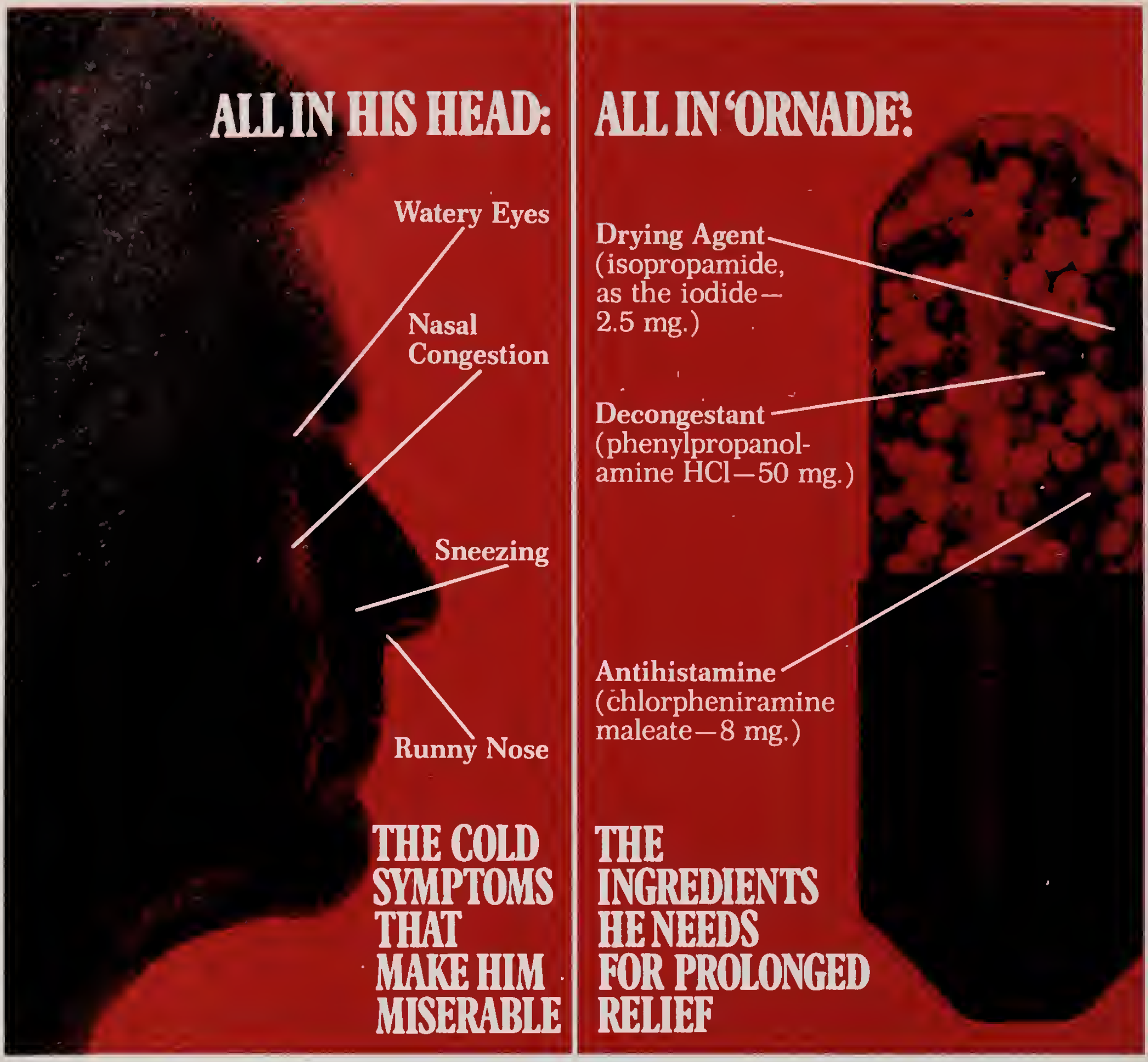
Through the day, every day,
ulcer patients take
one million doses of Mylanta
for relief of ulcer pain.

Mylanta®
LIQUID/TABLETS
aluminum and magnesium hydroxides plus simethicone

Good taste = patient acceptance
Relieves G.I. gas distress*
Non-constipating

*with the defoaming action of simethicone

Stuart PHARMACEUTICALS Pasadena, Calif. 91109
Division of Atlas Chemical Industries, Inc., Wilmington, Del 19899


ALL IN HIS HEAD:
Watery Eyes
Nasal Congestion
Sneezing
Runny Nose
THE COLD SYMPTOMS THAT MAKE HIM MISERABLE
ALL IN 'ORNADE':
Drying Agent (isopropamide, as the iodide—2.5 mg.)
Decongestant (phenylpropanolamine HCl—50 mg.)
Antihistamine (chlorpheniramine maleate—8 mg.)
THE INGREDIENTS HE NEEDS FOR PROLONGED RELIEF

Before prescribing, see complete prescribing information in SK&F literature or *PDR*.

Indications: Upper respiratory congestion and hypersecretion associated with the common cold; acute and chronic sinusitis; vasomotor rhinitis; allergic rhinitis (hay fever, "rose fever," etc.).

Contraindications: Hypersensitivity to any component; concurrent MAO inhibitor therapy; severe hypertension; bronchial asthma; coronary artery disease; stenosing peptic ulcer; pyloroduodenal or bladder neck obstruction. Children under 6.

Warnings: Advise vehicle or machine operators of possible drowsiness. Warn patients of possible additive effects with alcohol and other CNS depressants.

Usage in Pregnancy: In pregnancy, nursing mothers and women who might bear children, weigh potential benefits against hazards. Inhibition of lactation may occur.

Effect on PBI Determination and I^{131} Uptake. Isopropamide iodide may alter PBI test results and will suppress I^{131} uptake. Substitute thyroid tests unaffected by exogenous iodides.

Precautions: Use cautiously in persons with cardiovascular disease, glaucoma, prostatic hypertrophy, hyperthyroidism.

Adverse Reactions: Drowsiness, excessive dryness of nose, throat or mouth; nervousness; or insomnia. Also, nausea, vomiting, epigastric distress, diarrhea, rash, dizziness, weakness, chest tightness, angina pain, abdominal pain, irritability, palpitation, headache, incoordination, tremor, dysuria, difficulty in urination, thrombocytopenia, leukopenia, convulsions, hypertension, hypotension, anorexia, constipation, visual disturbances, iodine toxicity (acne, parotitis).

Supplied: Bottles of 50 capsules.

SK&F *Smith Kline & French Laboratories*

Trademark

ORNADE® SPANSULE®

Each capsule contains 8 mg. of Teldrin®(brand of chlorpheniramine maleate); 50 mg. of phenylpropanolamine hydrochloride; 2.5 mg. of isopropamide, as the iodide.

brand of sustained release capsules

UNCOMMON RELIEF FOR COLD SYMPTOMS

OR-203

ANNUAL BUSINESS MEETING
November 15, 1971

Agenda

1. Call to order—M. C. Korengold, M.D.
2. Memoriam to Deceased Members—All Rise
3. Remarks of the President—M. C. Korengold, M.D.
4. Remarks of the Chairman of the Executive Board—H. D. Ecker, M.D.
5. Confirmation of New Members
6. Nominations to Health Foundation
7. Report of the Nominating Committee—Election of Officers for 1972
8. Introductions of New Officers and Society Representatives
9. Adoption of Amendments to the Constitution and By-Laws
    - A) Chapter IV
    - B) Chapter V
    - C) Chapter VI
    - D) Chapter VII, Article 1.
    - E) Chapter VII, Article 3.
    - F) Chapter VIII, Article 1.
10. Adoption of 1972 Budget
11. Proposed Dues Scheduled for 1972
12. Introduction of D. C. Crain, M.D., President for 1972—M. C. Korengold, M.D.
13. Presentation of Gavel to M. C. Korengold, M.D., Outgoing President—H. D. Ecker, M.D.
14. Other Business
15. Adjournment

Reports

Report of the Chairman—Executive Board
Report of the Secretary-Treasurer
Report of the Board of Credentials
Report of the Woman's Auxiliary

In Memoriam

Ayers, M. Francis	Associate	November 30, 1970
Bartch-Dunn, Anna	Life	October 13, 1970
Beard, William	Active	July 2, 1971
Bruner, Weston	Active	September 8, 1971
Burns, Cornelius	Associate	April 3, 1971
Clark, Myles H.	Active	April 24, 1971
Cooper, C. David	Associate	April 8, 1971
Costello, James	Special	February 24, 1971
Douglas, Harry S.	Active	March 25, 1971
Gay, Lendall	Special	July 28, 1971
Hightower, Robert	Associate	April 18, 1971
Johnson, Stewart	Active	July 1, 1971

Jones, Benjamin	Active	June 18, 1971
Kelso, Richard	Associate	March 29, 1971
Kreuzburg, Franklin	Special	June 24, 1971
Kulesher, Henry	Associate	October 18, 1971
Kyle, Laurence	Active	April 24, 1971
Lattman, Isidore	Special	July 20, 1971
Manion, William	Associate	November 9, 1970
Markwood, Emmett	Active	June 27, 1971
McFall, Creed	Associate	November 22, 1970
Nathanson, Leon	Active	August 24, 1971
Pelzman, Ivy	Associate	December 25, 1970
Schwartzbach, Saul	Active	November 1, 1970
Shepherd, Elmer	Life	February 15, 1971
Sherburne, John	Associate	November 2, 1970
Smith, Leonard	Active	March 6, 1971
Tobin, William	Active	March 3, 1971
Watt, Mary F.	Active	November 27, 1970
Wood, Karl H.	Associate	August 3, 1971

New Members

Active

Advani, Mohan T.
Antonio, Jose Marco
Ayers, William
Beller, Robert F.
Berkman, Peter
Besselaar, Gerrit
Bisaccia, Leonard
Blemly, Nelson
Bradt, Jack O.
Caceres, Cesar
Calhoun, Thomas
Carlson, David
Chapman, Jr., Joseph C.
Chatel, John
Cheng, Tsung
Chidel, Ira
Citgay, Olcay
Coplon, Frederic
Corbin, Maurice C.
Cuevo, Nemesio G.
D'Angelo, James
de Moya, Armando
Dengel, Gisela
Dobbins, William O.
Donshik, Peter C.
Dowling, John A.
Dupont, Robert
Durell, Jack
Edelsburg, John
Edwards, Charles C.
Fermaglich, Joseph
Finkelor, Thomas M.
Flores, Prospero A.
Fortune, William
Gargour, Guy
Garrett, Margaret L.
Gerwin, Kenneth
Giere, Joseph
Giller, Martin
Groban, Stephen E.
Herman, Monte
Hoffman, Howard
Hou, Beinhard
Hunter, Allan A.
Iglehart, Vivian R.
Jacobs, William
Jensen, Wallace
Kaufman, Ronald
Kelly, John
King, Joseph D.
Kirschner, Leslie G.
Kish, Massih
Klein, Lawrence
Klotz, Edward
Kwass, Stephen
LaRosa, John

Latchis, Erika
Lemp, Michael
Levi, L. David
Lieberman, Norman A.
Lindgren, Keith M.
Liu, Chia-Ching
Lubar, Paul T.
Lux, Anna M.
Malone, Michael
Marshall, John H. L.
McFarlane, Maurice
McGowan, Larry
Missel, Jerome L.
Morowitz, David A.
Moshyedi, Ata-Ohlah
Naughton, John
O'Reilly, Sean
Patton, Genieean P.
Pavot, Alfred P.
Pieczenik, Steve R.
Piemme, Thomas
Polatin, Peter
Pooya, Manoochehr

Ramaswamy, Davanhalli
Reba, Richard
Restak, Richard
Robinson, Janice Y.
Rolle, Albert
Rosser, Pearl L.
Saini, Virender
Salsberry, Otto
Siegal, Larry
Snow, Paul
Sobhany, Manochehr
Stubenbord, John
Taylor, Halley B.
Tunell, William
Vasarhelyi, Laszlo
Vlalukin, Boris
Weber, William F.
Webster, John B.
Weiss, Harold
Williams, Henry
Williams, Robert H.
Wilson, Jr., Norman
Wilson, Joseph R.

Associate

Bertman, Martin
Blumenthal, Lester S.
Boswell, Thornton
Conant, Richard
Devairs, Dionisios
Dobbins, John
Dorn, Paul
Fairbanks, David
Furst, William K.
Helmly, Robert

Hsu, Chin-Chuan
Jackson, Francis
Khatri, Ibrahim
Latchis, Kenneth
Levin, Jack J.
Lopez, Lambreto
Lorge, Heinz J.
Robinowitz, Caroline
Williams, Ernest
Yim, Woong Soon

Resident-Intern

Barr, Jr., Norman L.
Caesar, Hugo
Cutler, Leonard

Linzau, Hean
Robinson, James
Smith, III, Henry J.

Tegtmeyer, Charles J.

Nominations to the Health Foundation

The Health Foundation's By-Laws stipulate that the Board of Directors (eleven) be named by the Medical Society. The Board must include the Society's President, President-Elect, Secretary-Treasurer, three other members of the Society and five additional members including a dentist, a pharmacist, and a registered nurse. It is recommended that the following be approved as members of the Health Foundation's Board of Directors for one year beginning January 1, 1972.

President–MSDC
President-Elect–MSDC
Secretary-Treasurer–MSDC
Edward Nicklas, M.D.
Russell M. Tilley, M.D.
Grace Matthews, R.N.
Mrs. Jane Yates
Renah Camlier
Wilfred Dudley, D.D.S.
Mr. John McHugh
Roland Scott, M.D.

Report of the Nominating Committee To All Voting Members—October 1, 1971

The Committee met on Monday, September 27, 1971 in the Medical Society Buliding, at which time the following slate of candidates, for the offices indicated, was selected by closed ballot. In accordance with the By-Laws of the Society, this ballot is hereby published to the voting membership.

President-Elect	Irving Burka, M.D.
1st Vice President	Warren Brill, M.D.
2nd Vice President	Robert Langevin, M.D.
Member, Executive Board	Marvin C. Korengold, M.D.
Member, Executive Board	Leslie Hedgepath, M.D.
(Appointed by the Executive Board to fill Dr. Bacon's unexpired term)	
Member, Board of Credentials	William Funderburk, M.D.
Member, Board of Credentials	Kenneth McCoy, M.D.
Delegate to AMA	John J. Hughes, M.D.
Alternate Delegate to AMA	James Boland, M.D.
Board of Trustees of Group Hospitalization	Lewis Biben, M.D.
Board of Trustees of Medical Service of D.C.	Charles E. Bickham, Jr., M.D.
Board of Trustees of Medical Service of D.C.	John A. Kenney, Jr., M.D.
Board of Trustees of Medical Service of D.C.	Edward W. Nicklas, M.D.

Nominating Committee—1971

Robert H. Parrott, M.D.
Russell M. Tilley, M.D.
Thomas E. Curtin, M.D.
Jack J. Rheingold, M.D.
Wesley M. Oler, M.D.
Albert J. Miele, M.D.
John J. Lynch, M.D.
Frank S. Pellegrini, M.D. (Chairman)

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Constitution and By-Laws Amendments

Agenda Item #9-A

Existing

Chapter IV–Dues & Assessments

Article 1,

Section 2. Active members shall pay dues of eighty-five dollars ($85) annually; except that recent graduates who have been in the active practice of medicine for less than three years shall pay for their first year thirty dollars ($30), for their second year forty dollars ($40), and thereafter eighty-five ($85.00).

Section 3. Special members shall pay dues of ten dollars ($10) annually.

Section 4. Associate members shall pay dues of thirty dollars ($30) annually.

Section 5. Intern-Resident members shall pay dues of five dollars ($5) annually.

Section 6. Student members shall pay dues of two dollars ($2) annually.

Proposed

Chapter IV–Dues & Assessments

Article 1,

Sections 2, 3, 4, 5, & 6 deleted and the following substituted:

Section 2. Dues shall be set on an annual basis by the Executive Board, with the approval of the membership.

Agenda Item #9-B

Existing

Chapter V–Officers & Their Duties

Article 1,

Section 1. The elected officers of this Society shall be a President, a President-Elect who shall assume the office of President the year following his election, a First Vice-President, a Second Vice-President, a Delegate or Delegates and an Alternate or Alternates to the House of Delegates of the American Medical Association. There shall also be a combined office of Secretary-Treasurer and Executive Director which shall be filled by a qualified person not necessarily a physician whose selection, appointment and contract of employment shall be the duty of the Executive Board.

Section 2. The President shall preside over meetings of the Society; call meetings as provided in Chapter III, Article 1; and shall perform such duties as may be imposed on him by the Society and in general as pertain to his office. He shall be a member ex-officio of the Executive Board, and all Councils and Committees, and with approval of the Executive Board, shall appoint all members of the various Councils and Committees.

Section 3. The President-Elect, First Vice-President, and Second Vice-President shall be members ex-officio of the Executive Board.

Section 4. The Vice Presidents shall, in the order of their rank, assume the duties of the President during his absence or inability to act.

Proposed

Chapter V–Officers & Their Duties

Article 1,

Section 1. The elected officers of the Society shall be the President; the President-Elect, who shall assume the office of President the following year. A Delegate or Delegates and an Alternate or Alternates to the House of Delegates of the American Medical Association. There shall also be a combined office of Secretary-Treasurer and Executive Director which shall be filled by a qualified person not necessarily a physician whose selection, appointment and contract of employment shall be the duty of the Executive Board.

Section 2. Unchanged

Section 3. The President, President-Elect and immediate Past President shall be members ex-officio of the Executive Board.

Section 4. The President-Elect shall assume the duties of the President during his absence or inability to act.

Agenda Item # 9-C

Existing

Chapter VI–Election Procedures, Terms of Office

Article 1,

Section 2. The President-Elect and Vice President shall be elected for terms of one year and shall be ineligible for reelection for the same office until two years have elapsed after the expiration of their term of office.

Chapter VI

Article III. One elective member of the Executive Board shall be elected annually to serve for the following four years. Elective members of the Executive Board shall be ineligible for reelection as elective members for one year after the expiration of the four year term.

Whenever a vacancy occurs in any office (not automatically filled) it shall be filled by the Executive Board until an election for the remainder of the term of office shall be held by the Society.

Proposed

Chapter VI–Elective Procedures, Terms of Office

Article 1,

Section 2. The President-Elect shall be elected for a term of one year and shall be ineligible for reelection for the same office until two years have elapsed after the expiration of his term of office as immediate Past President.

Chapter VI

Article III. Two elected members of the Executive Board shall be elected annually to serve for the following three years. Elected members of the Executive Board shall be ineligible for reelection as elected members for one year after the expiration of the three year term; but they shall be eligible for nomination as officers of the Society at any time.

Whenever a vacancy occurs in any office (not automatically filled) it shall be filled by the Executive Board until an election for the remainder of the term of office shall be held by the Society.

Agenda Item # 9-D

Existing

Chapter VII–Duties of the Executive Board

Article 1. The Executive Board shall consist of the President, the President-Elect, First Vice-President, Second Vice-President, and Delegates to the American Medical Association (all ex-officio); the Secretary-Treasurer, ex-officio without vote, and four elective members, one of whom shall be elected annually each year, to hold office for a term of four years. Six members of the Executive Board shall constitute a quorum for the transaction of business.

Proposed

Chapter VII–Duties of the Executive Board

Article 1. The Executive Board shall consist of the President; the President-Elect; the immediate Past President; and the Delegates to the American Medical Association, all ex-officio; the Secretary-Treasurer, ex-officio without vote; and six elected members, two of whom shall be elected annually to hold office for a term of three years. Members of the Executive Board elected prior to January shall serve for their full elected term. Six members of the Executive Board shall constitute a quorum for transaction of business. The immediate Past President shall serve as Chairman of the Executive Board.

Agenda Item #9-E

Existing

Chapter VII—Duties of the Executive Board

Article III,

Section 4. The Executive Board shall consider all disciplinary actions against members. Such action can be taken only after it has received a written report from the Grievance Committee and shall take action only after the time allowed in these By-Laws for an appeal has elapsed. A record of complaints, findings and other matters pertaining to the question involved, shall be kept.

A member against whom disciplinary action is being considered shall be so informed and notified that he may appear before the Executive Board on a given date, not less than four weeks from the date of the notice and present his defense. A member shall have ten days from the date the letter was mailed in which to notify the Secretary in writing that he wishes to appear before the Executive Board. Should the member not wish to appear, the Executive Board shall consider the case as presented in the record, together with any statement in writing which the member may present. When the Board has made its decision, the member shall be notified promptly. The Executive Board, by a seven-tenths vote of its total membership, may reprimand or expel a member or suspend any or all of his privileges.

Should he wish to appeal the decision of the Executive Board to the Society, he may do so by informing the Secretary in writing within ten days after the date the decision of the Board was mailed to him. In case of an appeal, and after the elapse of at least two weeks, during which the Secretary shall inform all Active, Life, and Special members of the details of the case, the Society may consider the appeal at any meeting.

Whenever a penalty has been levied against a member the Secretary shall notify the membership.

Proposed

Chapter VII—Duties of the Executive Board

Article III,

Section 4. The Executive Board shall consider all disciplinary actions against members. Such action shall be taken only after the Board has received a written report from the Grievance Committee, Committee on Medical Ethics and Professional Conduct or from whatever other duly constituted Committee of the Society which has reviewed the original complaint.

A member against whom disciplinary action is being considered shall be so informed and notified, by registered mail, that he may appear before the Executive Board on a given date, not less than four weeks from the receipt of the notice and present his defense. A member shall have ten days from the date the letter was mailed in which to notify the Secretary in writing that he wishes to appear before the Executive Board. Should the member not wish to appear, the Executive Board shall consider the case as presented in the record, together with any statement in writing which the member may present. When the Board has made its final decision, the member shall be notified promptly by registered mail. The Executive Board, a three-fourths vote of its total membership, may reprimand a member, suspend any or all of his privileges or expel him.

Should the member wish to appeal the decision of the Executive Board to the Society, he may do so by informing the Secretary in writing within ten days after the date the decision of the Board was received by him. In case of such appeal, and after the elapse of at least two weeks, during which the Secretary shall inform all Active, Life, and Special members that such appeal will be heard, the Society may consider the appeal at any meeting.

Whenever a penalty has been levied against a member the Secretary shall maintain as a portion of the minutes of the meeting of the Board during which the penalty is levied a record of the proceedings including the complaint, the defense presented, the findings of the Board and all other matters pertaining to the question involved.

Agenda Item #9-F

Existing

Chapter VIII–The Board of Credentials

Article 1. The Board of Credentials shall consist of six Active, Life or Special members, two of whom shall be elected each year for terms of three years. The Board shall perform its duties as outlined in CHAPTER II, Article VIII. The Chairman and Vice Chairman shall be appointed by the Executive Board. At least three members shall be present and voting at each meeting of this Board.

Proposed

Chapter VIII–The Board of Credentials

Article 1. The Board of Credentials shall consist of not less than six Active, Life or Special members, four of whom shall be elected each year for terms of three years. The Board shall perform its duties as outlined in Chapter II, Article VIII. The Chairman and Vice Chairman shall be appointed by the Executive Board. At least six members shall be present and voting at each meeting of this Board.

Proposed Budget
1972

Society Income*	
Active Dues	$235,085.00
Associate Dues	28,080.00
Resident-Intern Dues	480.00
Student Dues	375.00
Scientific Assembly Income	19,250.00
Regional Medical Program Reimbursement	81,000.00
Publication Income	67,727.00
Medical Bureau Income	299,086.00
Building Space Receipts & Reimbursements	93,624.00
Other Income (Interest, A.M.A. Rebate)	8,000.00
Gross Society Income	$832,707.00
Society Expenses	
Administrative Expense	$186,048.00
General Operations Expenses	79,944.00
Special Membership and Committee Activity	64,384.00
Scientific Assembly Expense	27,250.00
Membership Department Expense	13,794.00
Publications Expense	67,727.00
Medical Bureau Expense	288,728.00
Building Operational Expense	68,657.00
Mortgage Principal Curtailment	25,000.00
Total Society Expenses	$821,532.00
Net Income from all Functions	$ 11,175.00

* Income based on approval of new dues schedule as stated in Agenda Item #11

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Proposed Dues Schedule for 1972

Upon the recommendation of the Finance Committee, the Executive Board approved the following schedule of dues in order to support the Budget for 1972.

Active Membership	$145
Active Membership (1st Year Practice)	$45
Active Membership (2nd Year Practice)	$55
Special Membership	$25
Associate Membership	$40
Resident-Intern-Membership	$15
Student Membership	$5

Report of the Chairman—Executive Board

Medical Annals

With diligence and skill, an Ad Hoc committee of the Executive Board with Dr. William McCune as Chairman conducted a survey, including a questionnaire to the Society members, of our Scientific Journal, the MEDICAL ANNALS. As a result of the excellent response to the questionnaire a number of changes have materialized. Beginning January 1, 1972 the ANNALS will take on a new look under the direction of Dr. Raymond Scalettar, who was selected by the Board as Associate Editor.

Some of the changes approved by the Board deal with design and layout as well as with content. In addition to scientific articles, the ANNALS will carry expanded editorial comment, reports and articles on current socioeconomic affairs, and various other innovative articles.

Drug Abuse

Working closely with the Interdisciplinary Committee on Drug Dependency, President Korengold and Chairman Donald Glew have taken the Society literally to the forefront of the problem of drug abuse and drug dependency in this area as it relates to methadone. After considerable deliberation by the Committee during the earlier months of this year, the Committee developed and the Board approved specific guidelines for our physicians to adhere to regarding methadone detoxification and maintenance. Numerous meetings were held with various agencies which are also dealing with this drug problem. Cooperation was also solicited from the D.C. Pharmaceutical Association.

Because of its very diligent efforts, the Committee and its leadership have been successful in reducing the amount of illicit seepage of methadone to the suburbs and to the Baltimore area. More recently, upon the recommendation of the Committee, and with the cooperation of the D.C. Pharmaceutical Association, the Society declared a moratorium on all prescriptions for methadone.

Legislation

Through the past several years the Society, guided by the work of the Legislative Committee, has put enormous time and effort into several items of legislation which were introduced into Congress only to see them die with the adjournment of each session. Earlier this year, with the cooperation of the local AMA office, a legislative assistant was employed. Due to the efforts of this individual, significant progress has been made with moving three specific bills through Congress to the point where we can hope for passage of at least two of these before the end of this first session.

The Incorporation Bill H.R. 3121 has been passed by the House of Representatives and is now being considered on the Senate side. The Healing Arts Practice Bill H.R. 6119 was passed by the House and subsequently by the Senate, but only after the Senate had made major changes. The Bill is being prepared for deliberation by a Conference Committee of both Houses at which time it is hoped that an acceptable compromise will be reached. The Medical Records Act H.R. 9769 was introduced into the House by Representative Hagan shortly before Congress recessed for the summer. Hearings are being held at this time on this bill by the Education Subcommittee of the House District Committee.

Medical Foundation

One of the subjects of our time regarding the future delivery of health care is that of the Medical Foundation. So prominently has this mechanism been discussed that an association on a national level has been formed for "medical care foundation." An Ad Hoc Committee of the Board, under the Chairmanship of Dr. James Boland, has spent most of the year looking at the operations of various foundations from all over the country. In light of the findings of this Committee, the Board has authorized it to proceed with the incorporation document of a foundation for the Washington area. Considerable work remains to be done before a viable foundation can function. It is hoped that this Committee will have the broad support necessary to bring this objective to fruition.

Regional Medical Programs

In 1966 the Society applied, and received in 1967, granteeship of the regional medical programs. Throughout these six years many people have devoted tremendous effort and time to this program. Within the last 18 months new developments have taken place which should be reported to you. As you may know, Congress reduced funding for this program by 50% in this fiscal year, while at the same time, the program has veered from a categorically oriented program to one which is now directed toward delivery of health care and training of health manpower.

During this past year a Special Committee of the Board has analyzed these changes as they might affect the Medical Society. In line with these changes and the cut in funds in the Society's grant, the core staff which is housed in the Society building was augmented with the "project staff" which heretofore had been housed at the Hospital Council Headquarters. In mid-October a "management evaluation" visit was held and we can expect a "program" site visit in mid-December.

Personnel

Franc Ferraraccio was selected by the Executive Board to replace Mr. Thomas Sadler as Executive Director. In March 1971, Mr. Ferraraccio assumed full responsibility. He has served effectively and has demonstrated sincere interest as well as competent management of Medical Society affairs. Innovative ideas in many areas—organization, finance, publications, building maintenance, have characterized his approach as Director and they have improved the functions of the Society. I am confident, as the commercial of one of the insurance companies advertises, that "we are in good hands."

John Lewis Smith, III, as Counsel, represents a new look in Medical Society structure. Young, intelligent, interested, he provides reliable guidance and participates actively and personally in a wide range of our affairs. Frequent presence at Executive Board meetings maintains his familiarity with our problems as he engages in dialogue dealing with legal aspects of our operations. Constant contact together with personal participation by a genuinely interested lawyer has resulted in greatly improved management of related affairs, and has also engendered a satisfying sense of comfort and security in the members of the Executive Board. We hope that Mr. Smith will be with the Society for a long time.

Membership

1971 marked the advent of a "new look" at Society membership. Dr. Korengold's recommendation for dinner meetings was implemented, and judging from the attendance generated, received enthusiastic endorsement. Personal contact and participation of literally hundreds of members was achieved. It is hoped that this innovation will be made a regular event in future years.

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Student membership has increased many fold and mechanisms for more active student participation in the affairs of the Society are in the process of development and implementation.

Receptions at which new members can meet the officers of the Society are being held. This will undoubtedly serve to encourage prompt, active involvement by the physicians in Society programs and committee work.

Smoking and Health

Recognition is due this Committee for its vigorous action, through publicity, urging abstinence from cigarette smoking, and enlisting action by national airlines to provide separate sections in aircraft for smokers and nonsmokers. The Society publicly expressed its appreciation at our annual luncheon by awarding citations to four of these airlines lauding their action.

Constitution and Bylaws

Significant changes in the Constitution and ByLaws will be presented for approval by the Society this year. Greater representation on the Executive Board will be accomplished if these recommendations are endorsed. Other modifications in Society operations will serve to create a more effective and responsive organization. These changes reflect much thought and careful analysis by special committees. They deserve your attention. We hope they will be accepted for the betterment of the Society as a whole.

BC/BS Liaison

Several years ago the Executive Board approved a recommendation that regular semiannual reports be made at Board meetings by the Society physician member of the Board of Medical Service and GHI. This year, this practice has been activated and a closer liaison between these governing bodies has been created. Mutual interchange of information regarding policies, procedures and problems has thus, we believe, resulted in improved overall relationships. It is planned to continue this arrangement in the future.

Committees

Throughout the year 44 committees have performed a variety of services to our Society and the community at large. Each has earned commendation for a job well done. It is perhaps unfair to single out any special group, but it seems not inappropriate to mention particularly the work of the Grievance Committee, the Committee on Medical Ethics and Professional Conduct, the Utilization Committee, and the Fee Review Committee. They bear the brunt of our self-policing mechanisms, serving to preserve and maintain the image and the integrity of the Society and its members in the eyes of the public. In the face of a threat to the eminent position of the physician in today's society, the activity of these Committees assumes tremendous importance. They deserve special thanks for a thankless task well done.

Special Events

The screening clinics which were held in the Society building during Community Health Week in past years were replaced with highly successful symposiums this year. Hundreds of interested and knowledgeable people were invited and the attendance was exceptional. Earlier in the year a symposium on "Water Resources" was planned and conducted by Dr. Clifton Gruver, which was equally successful.

This year the Scientific Assembly was held in two days instead of the usual three days. The success of this event can be judged by the fact that the same number of registrants were recorded as were last year in three days. In addition, new events were introduced for the first time which were well-received Socio-Economic Subjects and a Prayer Breakfast.

Report of the Secretary-Treasurer

It is a privilege and indeed a pleasure to be writing this report as Executive Secretary of the Medical Society. During this first year, I have come to feel at home with members, officers, and staff, many of whom are old acquaintances.

One of my very first impressions was the tremendous tempo of activity; and throughout this activity I noticed a constant striving for change. The many issues which are facing medicine have spawned not only the activity but with it the need for change in philosophy, objectives, and meaning of the Society.

A survey of all the meetings held this year shows a marked increase over previous years. I shall not list these activities in deference to the report of the Chairman and the President.

I have been especially impressed by the dedicated work of the officers, committee chairmen, and committee members. Much of this work I have found is conducted in their private offices. It is one of my goals therefore, to provide the kind of administrative staff which will enable them to minimize the impact of society work on their private practice. We shall strive to help in any way possible.

This year has been marked by a number of changes in the Society staff. The departure of the Executive Director and the Assistant Executive Director at the same time represented a significant loss of management direction at one time. In order to assure continuity and cohesiveness, the staff was reorganized into five separate functional areas, with the head of each division reporting to the Executive Director. The five divisions, and the administrative head of each, are Building Management and Accounting Services, Mr. Michael Eason; Medical Bureau, Mrs. Dora Roggenkamp; Conference Services, Miss Beth Custer; Publications, Miss Judith Perruso and Secretarial Services, which is without a manager at this time. Concurrent with the organizational changes, a number of procedural changes have been instituted and others are still to be made.

Change has also taken place in the physical aspects of your Medical Society Building. Through the work of the House and Building Committee, a major overhauling of the mechanical equipment has been in progress throughout the summer months. In addition, the auditorium and third floor conference rooms have been redecorated, and the entire building is being repainted. Routine maintenance (mechanical and housekeeping) has been improved with an "In-House" staff, replacing an inefficient contract arrangement.

Earlier this year the auditor's report showed, and indeed the first quarter report revealed, that dues income would not be sufficient to offset expenses, and therefore reserves would have to be used. As Treasurer, I must report that this in fact occurred. In addition, a number of non-budgeted expenditures have occurred which have further aggravated the overall financial picture. It is imperative therefore that after six years since the last adjustment, a substantial dues increase should be authorized.

The organizational and procedural changes which were made last year in the Medical Bureau I believe have improved services. Overall operations however have continued to show significant financial loss. This cannot be sustained beyond the end of this fiscal year, so here again, a significant increase in subscriber rates is paramount.

This report would not be complete if I failed to recognize the efforts and support of the people around me. I consider my advent into the Society during the Presidency of Dr. Korengold to be singularly privileged, because it has been a distinctive year of leadership. I am grateful for the guidance and counsel of the Chairman of the Board, Dr. Ecker, during this period of change. I am deeply thankful for the loyalty and cooperation of the staff, without them these accomplishments would not have been possible.

medical annals of the district of columbia

vol. 41 no. 2 february 1972

Report of the Board of Credentials

Since the last Annual Business Meeting, the Board of Credentials has interviewed 215 applicants for membership; 153 for Active membership; 47 for Associate membership; and 15 for Resident-Intern membership. An unprecedented number of students have been given membership this year. Sixty-eight applications were processed and approved.

The membership of the Society as of October 1, 1971 is as follows:

Active	*Associate*	*Resident-Intern*	*Special*	*Life*	*Honorary*	*Student*	*Total*
1,661	723	33	35	166	33	68	2,719

Year	*Active Membership*	*Total Membership*
1971	1,661	2,719
1970	1,642	2,596
1969	1,651	2,513
1968	1,640	2,497
1967	1,595	2,440
1966	1,555	2,444
1965	1,518	2,421
1964	1,535	2,313
1963	1,532	2,275
1962	1,505	2,260

Respectfully Submitted,
Edward E. Ferguson, M.D., *Chairman*
Eugene Clark, M.D., *Vice-Chairman*
Edwin Parker, III, M.D.
William J. Jaffurs, M.D.
Charles Ireland, M.D.
Robert W. Langevin, M.D.

Report of the Woman's Auxiliary

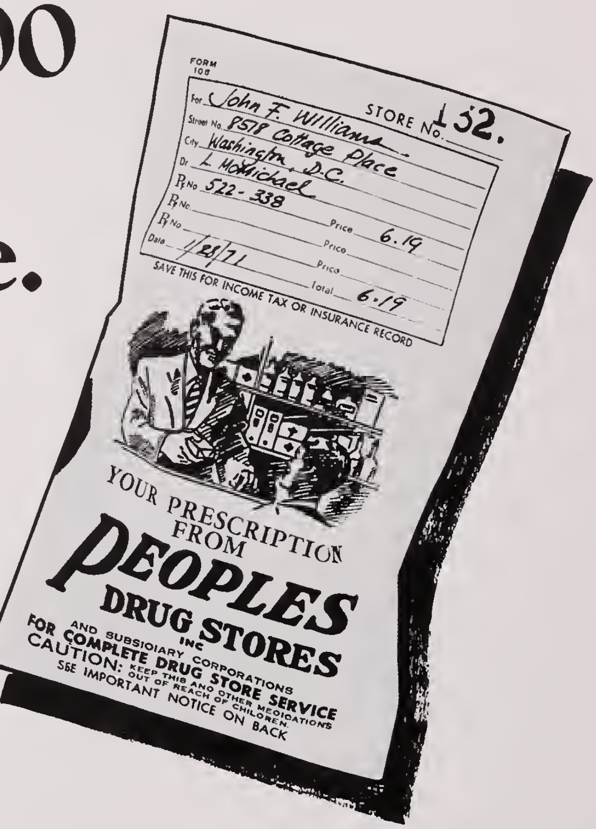
The Woman's Auxiliary to the American Medical Association celebrates its 50th anniversary this year and has set for its goal a national membership of 100,000 physician's wives. Our Auxiliary has responded to this appeal with 43 new members to bring our total membership to 403, the largest in our 46 year history. A new membership roster was printed this month to include all our new members. The get-acquainted reception sponsored by the President of the Medical Society for new Society members and their wives has been most helpful to us in our recruitment of new members. We invite every wife whose husband is an active or associate member of the Medical Society of the District of Columbia to join our Auxiliary and participate in our social activities, our fund raising projects for nursing scholarships and our pledge to the AMA-ERF student loan fund.

Our dues are still $10.00 for Active Membership and $5.00 for Associate Membership per year. If the physician-husband is a member of the AMA, we pay $4.00 per member to the national WA-AMA. This entitles us to send delegates to the annual convention, fall workshops and to obtain publications for program development.

The first Auxiliary sponsored Art Show, featuring works by our members, husbands and children will be held November 9, 1971 with a cocktail reception from 7 to 9 p.m. in the Exhibit Hall of the Medical Society Building. The show will also be open November 10th.

Mrs. George W. Hyatt
President, 1971-72

The bag that has earned more than 125,000,000 votes of confidence.



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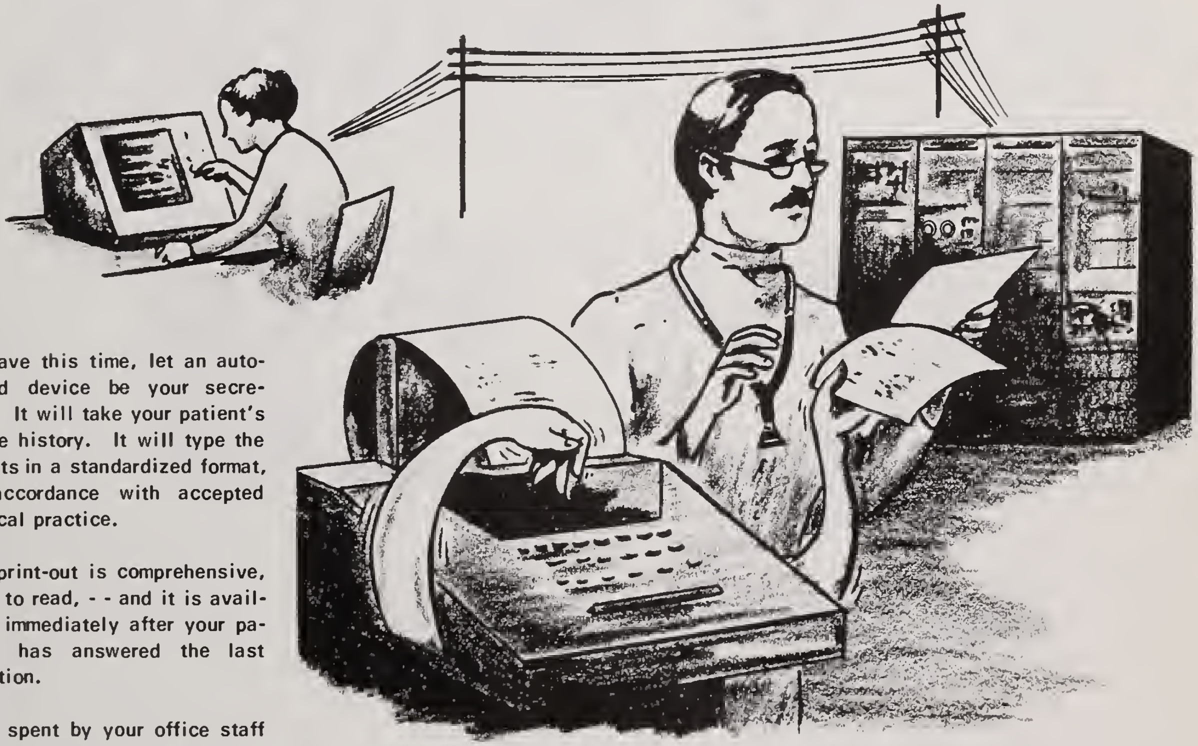

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Aunt Sarah, I love you.

Remember my 10th birthday, when you gave me that funny piece of green paper and everybody made such a fuss over it. (Except me.) Frankly, it was a pretty disappointing present for a kid. I couldn't eat it, couldn't play with it, couldn't wear it—and Mom took it away from me practically the minute I got it.

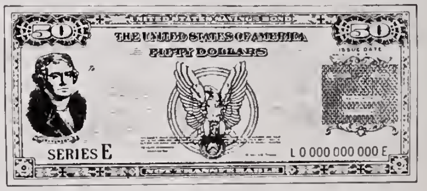
And every birthday after that (and Christmas, too), you gave me another one of those *Bonds*, you called them. I thought you were pretty gooney.

At least, I never had to guess what I was going to get from good old reliable Aunt Sarah.

And what I got was good old reliable U.S. Savings Bonds.

What I didn't realize then was that you gave me a gift of security for the future. Thanks to you and those Bonds, I had extra funds for college. And they helped when I bought my first new car.

Next week, I'm giving my nephew, Harry, his first U.S. Savings Bond. I know the kid is gonna think I'm some kind of nut. But, someday . . .



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Enhanced Absorption

Each tablet provides 115 mg elemental iron as the highly absorbable ferrous fumarate *plus* 600 mg of Vitamin C.

PHASE 2

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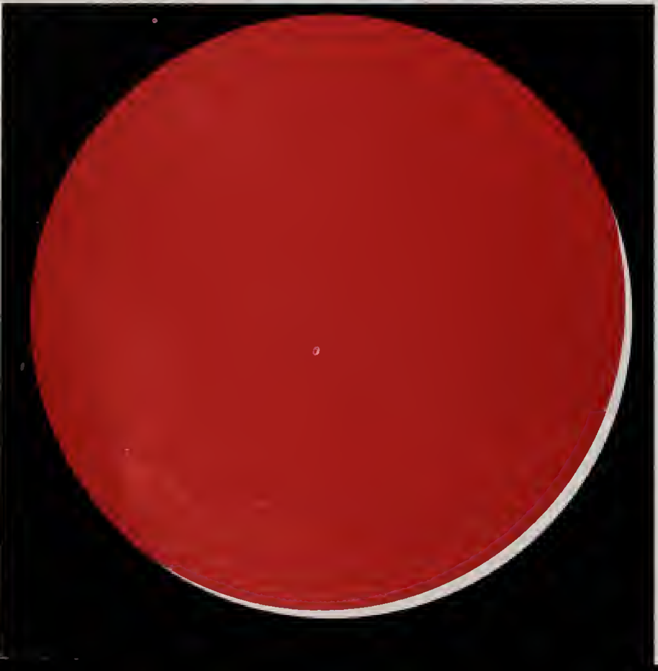
Each tablet provides Vitamin B_{12} (25 mcg) and Folic Acid (1 mg) to replace deficiencies.



PHASE 3

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Each tablet provides Vitamin E, which may be involved in lessening red blood cell fragility.

For common anemias
as well as problem ones

TriHEMIC™ 600

HEMATINIC TABLETS

Tri-Phasic Hematinic with 600 mg Vitamin C PLUS Vitamin E

Each tablet contains:

Vitamin C (Ascorbic Acid)	600 mg.
Vitamin B_{12} (Cobalamin Concentrate, N.F.)	25 mcg.
Intrinsic Factor Concentrate	75 mg.
Folic Acid	1 mg.
Vitamin E (*d*-Alpha Tocopheryl Acid Succinate)	30 Int. Units
Elemental Iron (as present in 350 mg. of Ferrous Fumarate)	115 mg.
Dioctyl Sodium Sulfosuccinate U.S.P.	50 mg.

Dosage: One Tablet Daily.
Available in Bottles of 30 Tablets.
On Your Prescription Only.

Precautions: Some patients affected with pernicious anemia may not respond to orally administered Vitamin B_{12} with intrinsic factor concentrate and there is no known way to predict which patients will respond or which patients may cease to respond. Periodic examinations and laboratory studies of pernicious anemia patients are essential and recommended. If any symptoms of intolerance occur, discontinue drug temporarily or permanently. Folic acid, especially in doses above 1 mg. daily, may obscure pernicious anemia, in that hematologic remission may occur while neurological manifestations remain progressive.

Adverse Reactions: G.I.: nausea, vomiting, diarrhea, abdominal pain. Skin rashes may occur. Such reactions may necessitate temporary or permanent changes in dosage or usage. Allergic sensitization has been reported following both oral and parenteral administration of folic acid.

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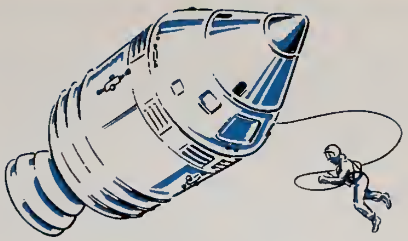

Man in space, now fait accompli, re-emphasizes the importance of Uro-Phosphate therapy. Research into the effect of space travel on the astronaut reveals that weightlessness causes loss of bone calcium. As the bones are required to bear less and less of the weight of the body they lose calcium, increasing the calcium content of the urine. When physical activity is reduced, the acidity of the urine should be adjusted to keep increased calcium in solution . . . . a prophylaxis to prevent kidney or bladder calculi.

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NOW A SUGAR-COATED TABLET

Each tablet contains: METHENAMINE, 300 mg.; SODIUM ACID PHOSPHATE, 500 mg.

Uro-Phosphate gives comfort and protection when inactivity causes discomfort in the urinary function. It keeps calcium in solution, preventing calculi; it maintains clear, acid, sterile urine; it encourages complete voiding and lessens frequency when residual urine is present.

Uro-Phosphate contains sodium acid phosphate, a natural urinary acidifier. This component is fortified with methenamine which is inert until it reaches the acid urinary bladder. In this environment it releases a mild antiseptic keeping the urine sterile.

Uro-Phosphate is safe for continuous use. There are no contra-indications other than acidosis. It can be given in sufficient amount to keep the urine clear, acid and sterile. A heavy sugar coating protects its potency.

Dosage:

For protection of the inactive patient 1 or 2 tablets every 4 to 6 hours is usually sufficient to keep the urine clear, acid and sterile.

2 tablets on retiring will keep residual urine acid and sterile, contributing to comfort and rest.

A clinical supply will be sent to physicians and hospitals on request.

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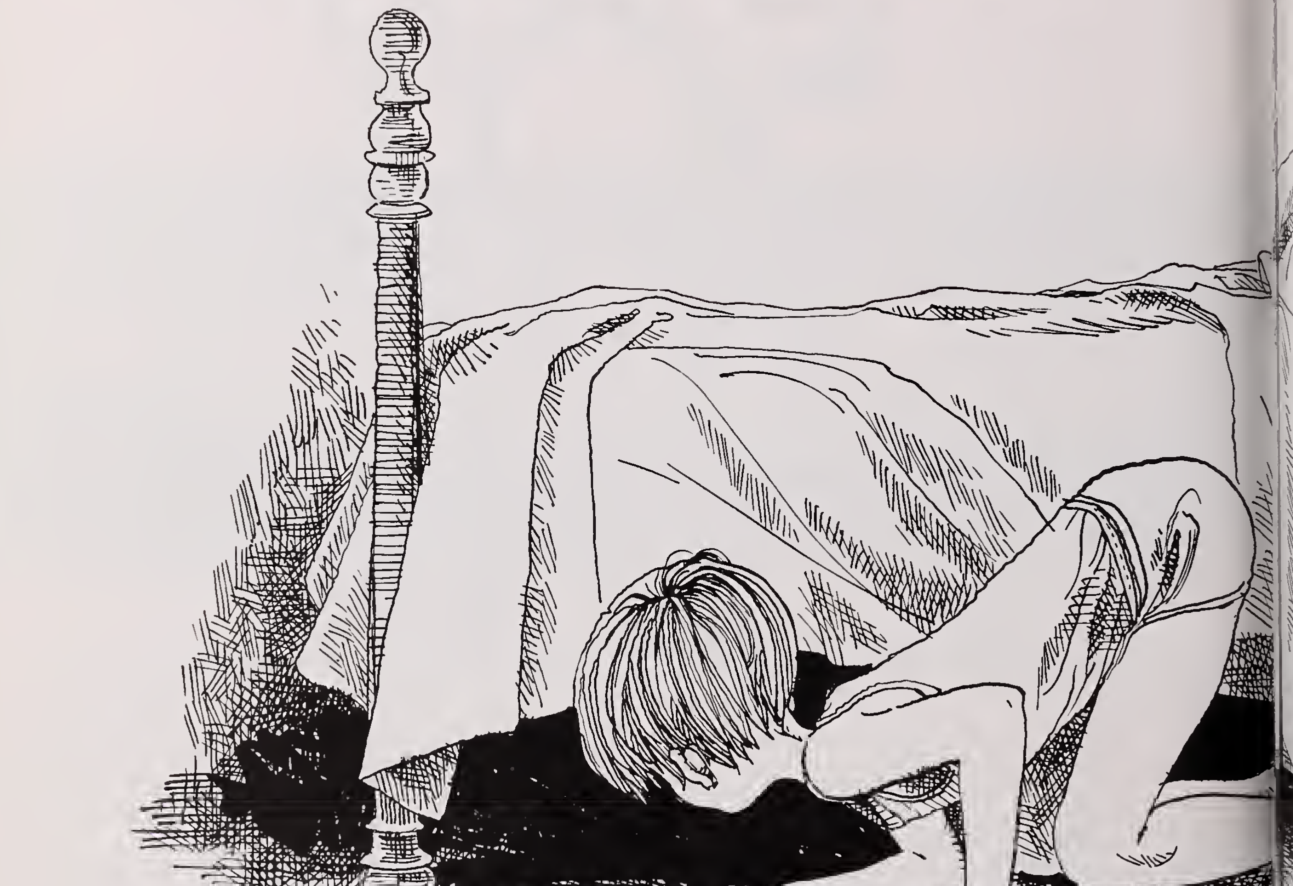

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Highly effective. Active against pinworm...and roundworm.

Non-staining. Doesn't stain teeth or oral mucosa on ingestion. Doesn't stain stools, clothing or linen.

Simple dosage. Single-dose regimen: 1 cc. per 10 lbs. of body weight (1 tsp. per 50 lbs.).

Well-tolerated. Based on pre-introductory studies.

Pleasant-tasting. Easy-to-take, caramel-flavored oral suspension.

Economical. One prescription for the entire family.

Antiminth (pyrantel pamoate) Oral Suspension

Actions. Antiminth (pyrantel pamoate) has demonstrated anthelmintic activity against *Enterobius vermicularis* (pinworm) and *Ascaris lumbricoides* (roundworm). The anthelmintic action is probably due to the neuromuscular blocking property of the drug.

Antiminth is partially absorbed after an oral dose. Plasma levels of unchanged drug are low. Peak levels (0.05-0.13 μg/ml) are reached in 1-3 hours. Quantities greater than 50% of administered drug are excreted in feces as the unchanged form, whereas only 7% or less of the dose is found in urine as the unchanged form of the drug and its metabolites.

Indications. For the treatment of ascariasis (roundworm infection) and enterobiasis (pinworm infection).

Warnings. *Usage in Pregnancy:* Reproduction studies have been performed in animals and there was no evidence of propensity for harm to the fetus. The relevance to the human is not known.

There is no experience in pregnant women who have received this drug.

Precautions. Minor transient elevations of SGOT have occurred in a small percentage of patients. Therefore, this drug should be used with caution in patients with pre-existing liver dysfunction.

Adverse Reactions. The most frequently encountered adverse reactions are related to the gastrointestinal system.

Gastrointestinal and hepatic reactions: anorexia, nausea, vomiting, gastralgia, abdominal cramps, diarrhea and tenesmus, transient elevation of SGOT.

CNS reactions: headache, dizziness, drowsiness, and insomnia.

Skin reactions: rashes.

Dosage and Administration. *Children and Adults:* Antiminth Oral Suspension (50 mg. of pyrantel base/ml.) should be administered in a single dose of 11 mg. of pyrantel base per kg of body weight (or 5 mg./lb.); maximum total dose 1 gram. This corresponds to a simplified dosage regimen of 1 cc. of Antiminth per 10 lbs. of body weight. (One teaspoonful 5 cc)

Antiminth (pyrantel pamoate) Oral Suspension may be administered without regard to ingestion of food or time of day; and purging is not necessary prior to, during, or after therapy. It may be taken with milk or fruit juices Because of limited data on repeated doses, no recommendations can be made

How Supplied. Antiminth is available as a pleasant tasting caramel-flavored suspension which contains the equivalent of 50 mg pyrantel base per ml., supplied in 60 cc. bottles.

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(pyrantel pamoate)
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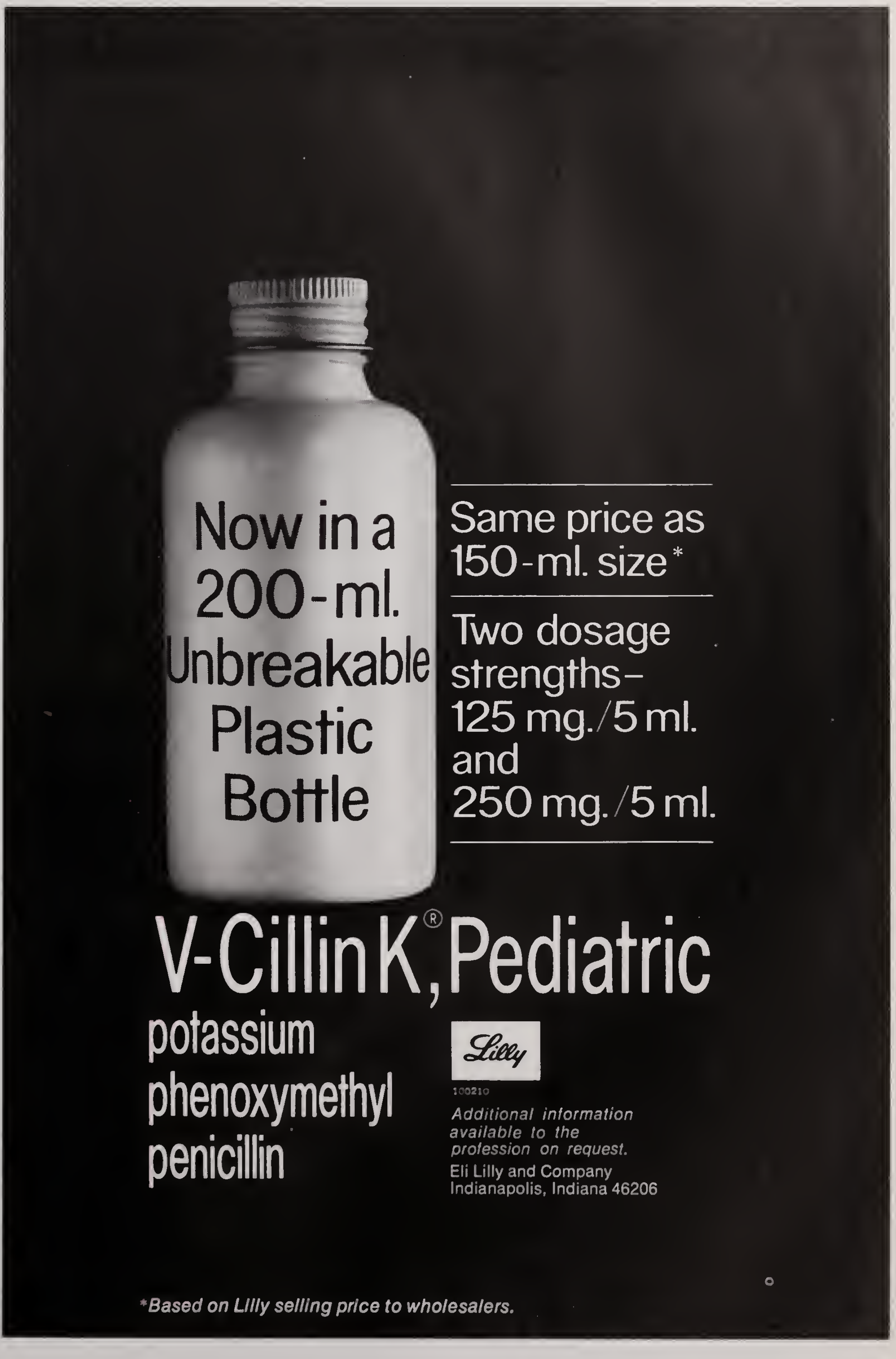

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125 mg./5 ml.
and
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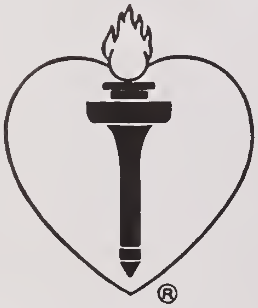

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Pre-Sate® (chlorphentermine hydrochloride)

Caution: Federal law prohibits dispensing without prescription.

Indications

Pre-Sate (chlorphentermine hydrochloride) is indicated in exogenous obesity, as a short term (i e several weeks) adjunct in a regimen of weight reduction based upon caloric restriction.

Contraindications

Glaucoma, hyperthyroidism, pheochromocytoma, hypersensitivity to sympathomimetic amines, and agitated states. Pre-Sate (chlorphentermine hydrochloride) is also contraindicated in patients with a history of drug abuse or symptomatic cardiovascular disease of the following types: advanced arteriosclerosis, severe coronary artery disease, moderate to severe hypertension, or cardiac conduction abnormalities with danger of arrhythmias. The drug is also contraindicated during or within 14 days following administration of monamine oxidase inhibitors, since hypertensive crises may result.

Warnings

When weight loss is unsatisfactory the recommended dosage should not be increased in an attempt to obtain increased anorexigenic effect; discontinue the drug Tolerance to the anorectic effect may develop. Drowsiness or stimulation may occur and may impair ability to engage in potentially hazardous activities such as operating machinery, driving a motor vehicle, or performing tasks requiring precision work or critical judgment. Therefore, such patients should be cautioned accordingly. Caution must be exercised if Pre-Sate (chlorphentermine hydrochloride) is used concomitantly with other central nervous system stimulants. There have been reports of pulmonary hypertension in patients who received related drugs.

Drug Dependence Drugs of this type have a potential for abuse. Patients have been known to increase the intake of drugs of this type to many times the dosages recommended. In long-term controlled studies with the high dosages of Pre-Sate, abrupt cessation did not result in symptoms of withdrawal.

Usage In Pregnancy The safety of Pre-Sate (chlorphentermine hydrochloride) in human pregnancy has not yet been clearly established The use of anorectic agents by women who are or who may become pregnant, and especially those in the first trimester of pregnancy, requires that the potential benefit be weighed against the possible hazard to mother and child. Use of the drug during lactation is not recommended. Mammalian reproductive and teratogenic studies with high multiples of the human dose have been negative.

Usage In Children Not recommended for use in children under 12 years of age.

Precautions

In patients with diabetes mellitus there may be alteration of insulin requirements due to dietary restrictions and weight loss. Pre-Sate (chlorphentermine hydrochloride) should be used with caution when obesity complicates the management of patients with mild to moderate cardiovascular disease or diabetes mellitus, and only when dietary restriction alone has been unsuccessful in achieving desired weight reduction. In prescribing this drug for obese patients in whom it is undesirable to introduce CNS stimulation or pressor effect, the physician should be alert to the individual who may be overly sensitive to this drug. Psychologic disturbances have been reported in patients who concomitantly receive an anorectic agent and a restrictive dietary regimen.

Adverse Reactions

Central Nervous System: When CNS side effects occur, they are most often manifested as drowsiness or sedation or overstimulation and restlessness. Insomnia, dizziness, headache, euphoria, dysphoria, and tremor may also occur. Psychotic episodes, although rare, have been noted even at recommended doses. **Cardiovascular:** tachycardia, palpitation, elevation of blood pressure. **Gastrointestinal:** nausea and vomiting, diarrhea, unpleasant taste, constipation. **Endocrine:** changes in libido, impotence. **Autonomic:** dryness of mouth, sweating, mydriasis. **Allergic:** urticaria. **Genitourinary:** diuresis and, rarely, difficulty in initiating micturition. **Others:** Paresthesias, sural spasms.

Dosage and Administration

The recommended adult daily dose of Pre-Sate (chlorphentermine hydrochloride) is one tablet (equivalent to 65 mg chlorphentermine base) taken after the first meal of the day. Use in children under 12 not recommended.

Overdosage

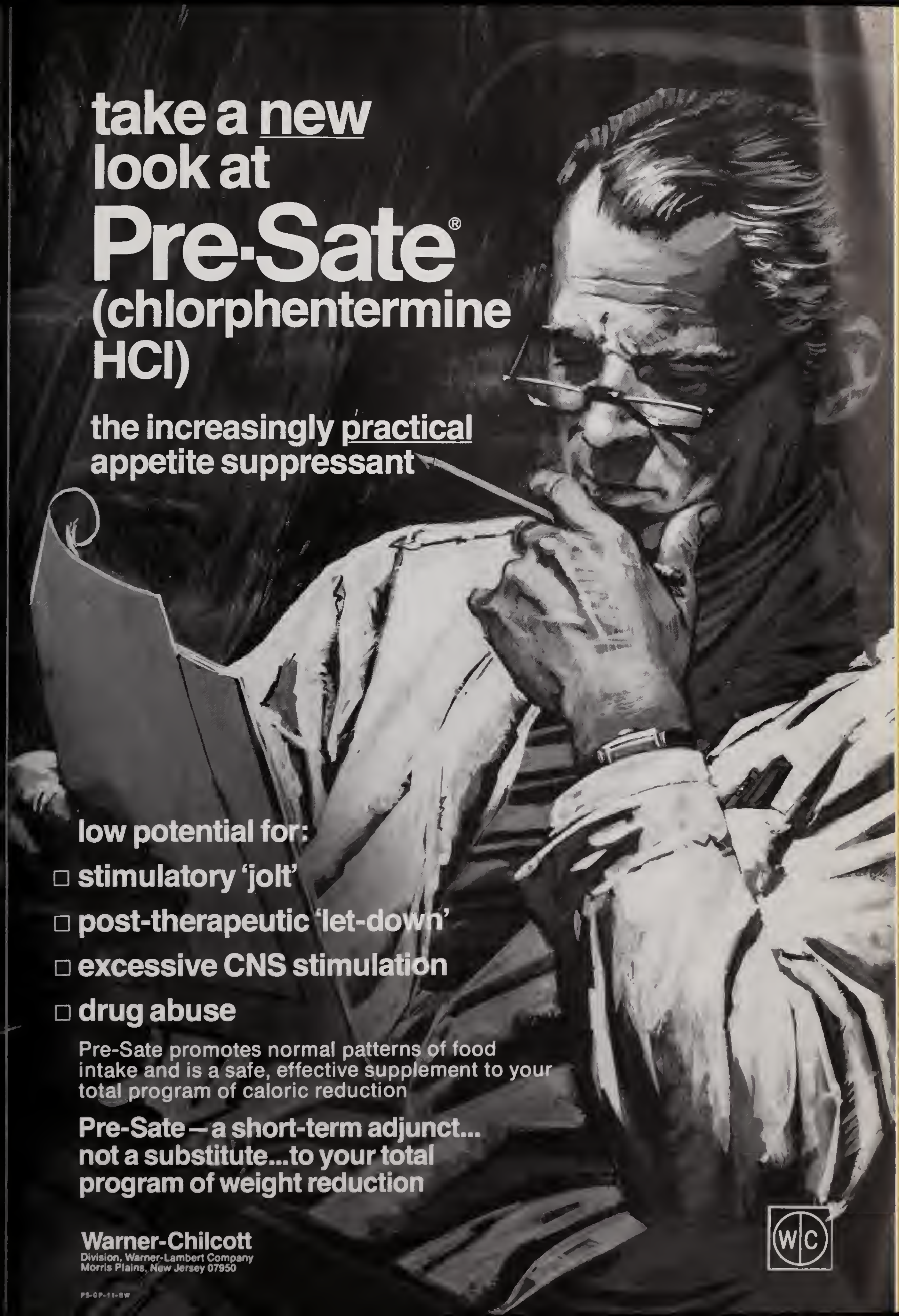
Manifestations: Restlessness, confusion, assaultiveness, hallucinations, panic states, and hyperpyrexia may be manifestations of acute intoxication with anorectic agents. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension, or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Fatal poisoning usually terminates in convulsions and coma.

Management: Management of acute intoxication with sympathomimetic amines is largely symptomatic and supportive and often includes sedation with a barbiturate. If hypertension is marked, the use of a nitrate or rapidly acting alpha-receptor blocking agent should be considered. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendations in this regard.

How Supplied

Each Pre-Sate (chlorphentermine hydrochloride) tablet contains the equivalent of 65 mg chlorphentermine base; bottles of 100 and 1000 tablets.

Full information is available on request.

take a new look at Pre-Sate®
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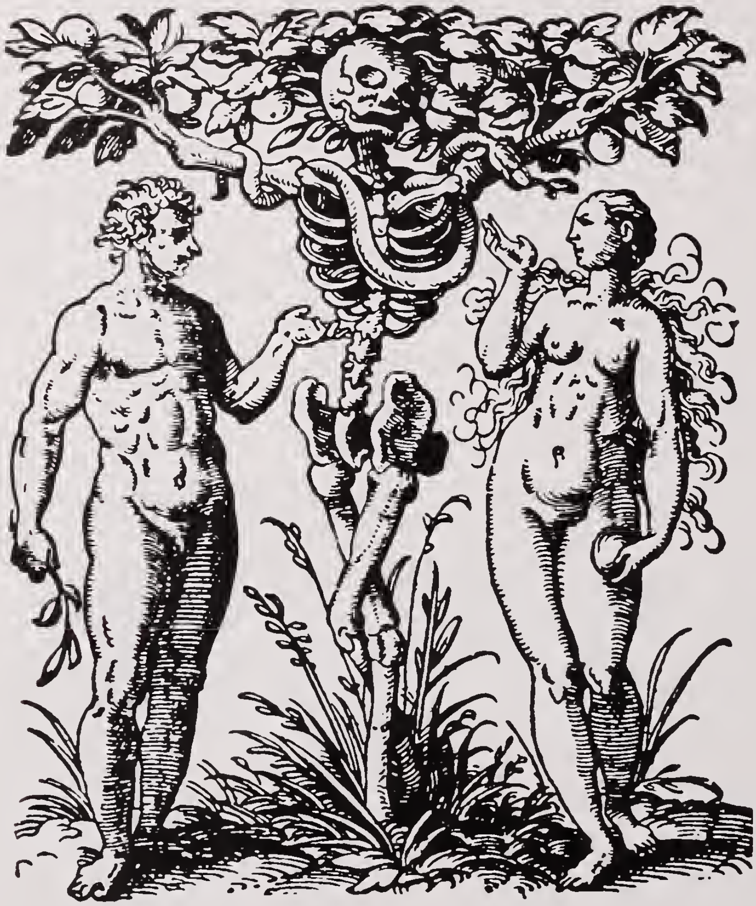
In acute gonorrhea

(urethritis, cervicitis, proctitis when due to susceptible strains of N. gonorrhoeae)



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Indications: Acute gonorrheal urethritis and proctitis in the male and acute gonorrheal cervicitis and proctitis in the female when due to susceptible strains of *N. gonorrhoeae*.

Contraindications: Contraindicated in patients previously found hypersensitive to Trobicin. Not indicated for the treatment of syphilis.

©1972 The Upjohn Company

Warnings: Antibiotics used to treat gonorrhea may mask o delay the symptoms of incubating syphilis. Patients should b carefully examined and monthly serological follow-up for a least 3 months should be instituted if the diagnosis of syphilis suspected.

Safety for use in infants, children and pregnant women has n been established.

Precautions: The usual precautions should be observed wit atopic individuals. Clinical effectiveness should be monitored t detect evidence of development of resistance of *N. gonorrhoea*

Adverse reactions: The following reactions were observe during the single-dose clinical trials: soreness at the injection sit urticaria, dizziness, nausea, chills, fever and insomnia.
During multiple-dose subchronic tolerance studies in norm human volunteers, the following were noted: a decrease in hemo

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single-dose intramuscular treatment

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gh cure rate:* 96% of 571 males, 95% of 294 females
(sages, sites of infection, and criteria for diagnosis and cure are defined below.)**

---

surance of a single-dose, physician-controlled treatment schedule

---

allergic reactions occurred in patients with an alleged history of penicillin sensitivity hen treated with Trobicin, although penicillin antibody studies were not performed

---

tive against most strains of Neisseria gonorrhoeae in vitro (M.I.C. 7.5-20 mcg/ml)

---

single two-gram injection produces peak serum concentrations averaging about 0 mcg/ml in one hour (average serum concentrations of 15 mcg/ml present 8 hours after dosing)

---

ote: Antibiotics used in high doses for short periods of time to treat gonorrhea may mask or delay the mptoms of incubating syphilis. Since the treatment of syphilis demands prolonged therapy with any ective antibiotic, and since Trobicin is not indicated in the treatment of syphilis, patients being treated for norrhea should be closely observed clinically. Monthly serological follow-up for at least 3 months should instituted if the diagnosis of syphilis is suspected. Trobicin is contraindicated in patients previously found persensitive to it.

---

ta compiled from reports of 14 investigators. **Diagnosis was confirmed by cultural identitication of *N. gonorrhoeae* on Thayer-rtin media in all patients. Criteria for cure: negative culture after at least 2 days post-treatment in males and at least 7 days post-atment in females. Any positive culture obtained post-treatment was considered evidence of treatment failure even though the low-up period might have been less than the periods cited above under "criteria for cure" except when the investigator determined t reinfection through additional sexuol contacts was likely. Such cases were judged to be reinfections rather than relapses or lures. These cases were regarded as non-evaluatable and were not included. JA72 1848-6

---

bin, hematocrit and creatinine clearance; elevation of alka- phosphatase, BUN and SGPT. In single and multiple-dose dies in normal volunteers, a reduction in urine output was ed. Extensive renal function studies demonstroted no con-ent changes indicative of renol toxicity.

sage and administration: Keep at 25°C and use within hours after reconstitution with diluent.
le—single 2 gram dose (5 ml) intramusculorly. Patients with orrheal proctitis and patients being re-treated after failure previous ontibiotic therapy should receive 4 grams (10 ml). In graphic areas where antibiotic resistance is known to be pre-ent, initial treotment with 4 grams (10 ml) intramuscularly is ferred.
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Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances, syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

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PRESIDENT'S MESSAGE

"Wisdom is the principal thing;
therefore get wisdom; and with all
thy getting, get understanding."

Solomon

"Knowledge comes; but wisdom lingers."

Tennyson

FEDERAL REGULATIONS NOW REQUIRE ALL PHYSICIANS TO BE RELICENSED BY WRITTEN EXAMINATION EVERY THREE YEARS.

Darrell C. Crain, M.D.

A rather grim headline - but one which some of our more liberal planners would like to see our newspapers feature.

Whether it be knowledge, wisdom, understanding or just plain medical information, the need for continual "getting" is being made more and more apparent to our profession by outside sources as well as by our peers. Surely this is one more example of the changing attitudes of our times. A few years ago it was assumed that the longer a man practiced, the more capable he was in the treatment of disease. "Experience is the best teacher" was sufficient to indicate that the kindly old family doctor sitting at the patient's bedside with the family standing in awe nearby, was the epitome of all medical knowledge and scientific acumen. And, as a matter of fact, he probably was.

But - no more! Today's practicing physician has only to go onto the wards of a university hospital and engage the first year intern or junior resident in conversation to learn how the recent graduate feels about his senior of five or more years. Even more significant is the increasing interest of laymen and legislators (the "consumer groups" - to use modern terminology) in assuring that doctors keep abreast of current developments.

In this regard, physicians in the Washington area are most fortunate for we practice in an area where there is ready access to the latest scientific developments. The bulletin circulated by the Metropolitan

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Washington RMP lists over 100 lectures, seminars and conferences, conducted monthly by teachers and leaders of our medical community.

But the mere fact that such conferences are being conducted - and indeed, are well attended - does not indicate that all physicians are taking advantage of them. Yet they must, and it is the responsibility of the Medical Society to assist them in doing so. I have therefore asked Dr. Jay Shapiro and his Committee on Continuing Medical Education to outline minimum requirements each physician should regularly fulfill to indicate he is adding properly to his medical knowledge. The task is not an easy one - each specialty has its own peculiar needs - and the implication of compulsion is odious to us all. Yet twenty-three states have already initiated such programs. In New Mexico, evidence of continuing education is now a requirement for the maintaining of a physician's license. In Arizona, Massachusetts, Oregon and Pennsylvania, it is a requirement for medical society membership. Recently, the Oregon Medical Society suspended eleven of its members for failing to comply with their Continuing Medical Education requirements. Eighteen other states, like the District of Columbia, have the matter under serious consideration. In most areas completion of the A.M.A. "Physician's Recognition Award" is adequate. A few physicians in our area have already qualified, many others are eligible, and need only complete the small amount of paper work to receive the award certificate. I suggest all members examine the requirements closely, undergo a period of self-assessment and then set up an individual program to correct shortcomings. Hopefully, then if some demonstration of our ongoing education is necessary, we will be prepared.

Darrell C. Crain

A MODERN PLAGUE

Webster defines "plague" as "that which smites or troubles . . . any afflictive evil; a scourge, an infestation". Until recently the plagues which doctors bent their efforts to prevent and treat were those caused by pathogenic microorganisms, and they were to be combatted by whatever was available in the medical armamentarium and by public health measures such as by drying up the breeding places of malaria-causing mosquitoes. But now we must deal with other plagues which are social rather than bacterial. One of these, which has reached epidemic proportions is a plague of guns responsible for the fearsome death toll among our citizens of 6,00 per year in the form of homicides alone as well as 47 per cent of all completed suicides. And if we are going to be effective in dealing with this plague we must employ other means than those which eradicated malaria.

Some myths about firearms which nurture the intensely felt opposition to the kind of gun control legislation which would begin to end this plague of guns have been exposed in a recent article in the American Journal of Psychiatry by Judge George Edwards of the U.S. Court of Appeals for the Sixth Circuit and a member of the National Commission on Reform of Federal Criminal Laws. Judge Edwards points out that the large majority of murders are the product not of cupidity, but rather of uncontrolled emotion in the course of quarreling among relatives or friends, quarrels that "might well result in nothing more than bloody noses or a lot of noise if there were not present a deadly weapon -- handy and loaded". This statistic is supported and refined in another article on the subject by Dr. Emanuel Tanay, a psychiatrist and lawyer, who describes most murders as resulting from the combination of a state of consciousness temporarily altered and impaired by psychopathological or chemical factors, and a handy weapon, usually a gun. The contention that our citizens need guns to protect themselves from criminals is vitiated not only by the above disclosure, but also by the fact that the police unanimously counsel against attempting to resist robbery because to do so is generally useless and dangerous. Actually there are far more innocent victims of accidents with guns than there are dead robbers killed by householders defending themselves.

The National Commission on Reform of Federal Criminal

Laws has recommended that Congress:

1) ban the production and possession of, and trafficking in handguns, with exceptions only for military, police, and similar official activities, and

2) require registration of all firearms.

The medical profession has a duty and responsibility to fight the plagues which afflict mankind, whatever their source or their nature. It is time for us to use our influence as guardians of the public health to support these sensible recommendations which ultimately will save lives. Accordingly, the Committee on Public Health of the Medical Society of D.C. has submitted to the Executive Board a resolution asking that the Society and the A.M.A. go on record as supporting, and actively support the enactment on a national level of gun control legislation.

Paul Chodoff, M.D.
Chairman
Mental Health Committee

THE DISTRICT OF COLUMBIA HOSPITAL CONFERENCE OF 1972

Health care is a right, not a privilege. Many of the medically indigent residents of the District of Columbia have in recent times come under third party insurance coverage, most notably Medicaid. These and other factors have brought citizens of the District who do not have private physicians to the Emergency Departments of various Washington Hospitals as their means of entering the health care delivery system. The number of patients increases monthly, hospital bed shortages have become more disparate, and the transfers of patients to D.C. General Hospital has continued to rise, to the dissatisfaction of patients, their families, and physicians at both the sending and receiving institutions.

Against this background of mounting pressure, discussions began between various representatives of the private community hospitals and D.C. General Hospital. It was decided that this represented a total community problem and should therefore be attacked by the total professional community. Neither D.C. General Hospital alone, nor the Department of Human Resources alone, are in a position to effect meaningful programs to provide adequate health care to this growing patient population. Accordingly, medical administrative and trustee representatives of all hospitals in the District of Columbia were invited to attend a preliminary plenary meeting - entitled "The District of Columbia Hospital Conference of 1972" - held on January 27, 1972. At this meeting, the problems of medical management and disposition of the acutely ill patients in emergency departments, ambulance transportation, and the transfer of such patients were discussed and defined.

Four working subcommittees were established under the following titles: 1) Committee on Indigent Bed Distribution; 2) Committee on Emergency Departments and Patient Transfer; 3) Committee on Ambulance Services; and 4) D.C. General Hospital Program Report. The Committee on Indigent Bed Distribution has been charged with developing recommendations for a distribution of indigent beds in all hospitals of the District, to develop an equitable formula for such a bed distribution, and to integrate such a plan with existing outpatient facilities, emergency departments, and ambulance services. The Committee on Emergency Departments and Patient Transfer has been charged with reviewing all aspects of emergency care and transfer of indigent patients, and to develop firm medical and administrative guidelines regarding patient transfers between any two hospitals.

The Committee on Ambulance Services has been charged with a review of existing emergency ambulance services and to make recommendations for: 1) integration of the services into an expanded transfer system, and 2) improvement of existing recruiting and training procedures for ambulance personnel. The Committee for the D.C. General Hospital Report consists of a small subcommittee charged with meeting with representatives of D.C. General Hospital to receive their report on problems, such as bed utilization, professional services, etc., and to forward their proposed solutions to the next meeting of the entire Conference.

Representatives of the Medical Society and the Medico-Chirurgical Society of D.C. are participating in this Conference. Mr. Joseph Yeldell, Director of the Department of Human Resources, has approved the concepts and goals of this Conference. The various working Committees begin work within the next few weeks, and reports of all Committees must be completed for the reconvening of the total Conference Ninety days after its initial meeting.

Will this Conference succeed in accomplishing anything? Why is this Conference any different from prior attempts to resolve these problems? To begin with, this is the first time that medical, administrative, and trustee representatives of all D.C. hospitals have gathered to consider these problems. Secondly, there has been no previous unified attempt to improve these areas under discussion originating from the responsible purveyors of services, and it has been apparent for some time that the D.C. Department of Health could not provide leadership for implementing such new programs. For the first time, representatives of the public hospital (D.C. General) sit at the conference table with representatives of the private community hospitals. Finally, with growing pressures from federal agencies and the Congress to provide adequate health care services to all people, it is extremely timely that the providers of services take the initiative to devise programs which will widen the services with increased efficiency, and hopefully, reduce the continuing cry for more and more funds. Should this Conference succeed in its goals to provide effective plans for improved indigent in-hospital and emergency department care in the District of Columbia, a similar forum might be convened at a later date, addressing itself to the problems of delivering outpatient services with greater breadth of coverage, improved efficiency and quality, and firm and balanced relationships to the in-patient services of the District Hospitals.

Louis Gillespie, Jr., M.D.

IN PURSUIT OF EXCELLENCE

On March 23rd, the Washington Hospital Center will open to patients a seven-unit wing specializing in coronary, medical, surgical, renal, respiratory, burn and psychiatric intensive care. As the largest, most modern wing of its kind, the new facility should make a significant contribution to the health care of the region.

In the coronary unit, for example, the master monitoring console is capable of providing continuous or emergency monitoring of patients from other areas of the hospital as well as from other hospitals. Through the use of data-phones, the electrocardiogram from a patient may be sent from another hospital to the central station where it can be displayed and observed for irregularities on a true-time basis. In addition, through the use of telecopiers, previously recorded electrocardiograms can be sent over the telephone lines to provide added information or consultative services concerning a particular patient.

In the renal unit, facilities for handling both acute and chronic renal and electrolyte problems have been included. The chronic hemodialysis program has the capacity to accommodate 18 chronic dialysis patients. New patients will be accepted in the program after evaluation following referral by a private physician.

The renal and respiratory units share some of the same facilities which will be advantageous in view of the considerable overlap in metabolic and acid-base problems manifested by both types of patients. The respiratory care unit will have a small surgical suite for such procedures as tracheostomy and bronchoscopy, so that these can be performed more rapidly, efficiently, and under optimum conditions. Because of the close proximity of the respiratory care unit to the pulmonary function laboratory, prompt determination of arterial blood gases will be assured. A radiographic room has been provided within this part of the intensive care wing which will further contribute to prompt diagnostic procedures, including angiography.

Patients with acute pre- and post-op surgical diseases will be accommodated in the surgical unit. Specifically, facilities for severe trauma cases, patients with shock of varying etiologies, post open-heart surgery patients, and organ transplantation patients have been provided.

Of particular significance to the region will be the Hospital Center's burn unit. It has its own treatment area for dressing changes and debridement. Hydrotherapy will also be a feature of the self-contained unit.

In the wing, both medical and surgical house-staff coverage will be provided on a 24-hour basis. A section of Acute Care Education has been created with the responsibility of training allied personnel. Teaching of intensive care has been a cooperative venture between physicians and nurses, and on-going educational programs are underway.

Concern for the emotional as well as the physical well-being of patients is reflected in the approach to intensive care utilized in the new wing. A multidisciplinary approach will incorporate input from the Department of Psychiatry in every service. When this approach is combined with recent technological advances in monitoring and communications, the medical and nursing staff will be equipped to enhance total intensive care, both at the Washington Hospital Center and at area hospitals which wish to participate.

Leon Gerber, M.D.
Chairman, ICU Committee
Washington Hospital Center

THE MEDICAL ADVISORY COUNCIL - CONCERNED PHYSICIANS

On March 13, 1956, the Board of Trustees of Medical Service of D.C. recommended that a section be added to the appropriate ByLaw establishing a Medical Advisory Committee, to be composed of one member from each organized medical group in the area; the Committee to "advise and confer with the Board of Trustees on fees and related matters".

The original Committee was to be composed of not less than eleven or more than twenty-five members - essentially representing the practicing physicians. Accordingly, 21 selected groups were invited to appoint a representative to the newly established Committee, and the first meeting was held on June 6, 1956. Subsequently, on October 8, 1963, the Board voted that the President of Medical Service of D.C. be authorized to appoint the Chairman, Vice-Chairman and Secretary of the Medical Advisory Committee from the Board of Trustees.

The initial topics presented to this original Committee consisted principally of fee recommendations, service coverage, surveys of local statistics and experience, etc.

It is interesting to note in past records, that many items about which recommendations were made in past years by former Committees, appear on current Medical Advisory Council agendas, i.e., on April 9, 1958, the Chairman of the Medical Advisory Committee proposed a subcommittee to study methods of obtaining a separate charter for Medical Service of D.C. in order to make it independent of Group Hospitalization of D.C., Inc. Today, the Medical Advisory Council is still pursuing, in essence, this proposal. On February 26, 1958, the Medical Advisory Committee recommended "Insurance Committee of the D.C. Medical Society to certify the reasonableness of physician charges" under the Major Medical Program; on August 11, 1970, the Board of Trustees accepted the Medical Advisory Council's proposal that final "peer review" of a physician's services and charges, etc., remain with his local Medical Society prior to the issuance of the Medical Advisory Council's approval of the proposed UCR Program of Medical Service of D.C.

On April 11, 1970, the Medical Advisory Council's recommendation that it elect its own officers from its membership was accepted by the Board of Trustees by way of an amendment to the ByLaws of Medical Service of D.C.

Now the Medical Advisory Council (its name having been changed by the Board of Trustees' action on June 26, 1963 from Medical Advisory Committee) is composed of 32 members representing 27 Specialty Sections of the Medical Society of D.C. and 5 Metropolitan Area Medical Societies, these members representing the area practicing physicians.

Current Medical Advisory Council agenda topics include, in addition to the changing of the Articles of Incorporation of Medical Service of D.C., the status of the National Association of Blue Shield Plans Joint Contract Administration Committee, recent unfavorable newspaper publicity, status of the external independent audit of Medical Service of D.C., concurrent care services and charges, basis of determining relative values, utilization of new forms, and computerized billing by Medical Service of D.C.

It appears that Metropolitan Area practicing physicians have yet another mechanism to guide the development of local health care standards and insurance coverage, utilization, and peer review; that such opportunity is still very much available, only requiring each individual physician's acceptance of his responsibility of being informed, of selecting accurate representation, and of actively supporting his beliefs and goals.

Mary Lou Barker, M.D.

GOVERNMENT REGULATION OF HEALTH CARE DELIVERY

Attempts are presently being made by a number of professional groups to ward off the federalization of the Health Care Delivery System by voluntary attempts at self regulation. Notably, these include the efforts of the Joint Commission on Accreditation of Hospitals through the revised manual for accreditation of hospitals. Interestingly enough, the JCAH has accepted the requirements and standards of performance for laboratories recently enacted under federal laws.

Today, it is a fact of medical life that the federal government is regulating the practice of laboratory medicine through PL 89-97 (Medicare) and PL 90-174 (Interstate Commerce). The District of Columbia comes under both acts, whereas Maryland and Virginia are covered under Medicare. Eventually, these laws will have regulatory effect on how other physicians practice. Furthermore, there need not be any additional legislation, merely the administrative adoption of revised regulations by the executive branch of the federal government. This mechanism of governmental control by administrative rules probably heralds the modus operandi by which HEW works out its control over the medical practices of internists, pediatricians, certain general practitioners, and any other physician who operates a laboratory within the confines of his office regardless of his expertise.

Specifically, these two laws and their codes of regulations are:

1. PL 90-174, the Clinical Laboratories Improvement Act of 1967 (CLIA) and its Code of Federal Regulations Title 42, Chapter 1, Part 74: Regulations for Licensure of Clinical Laboratories (Interstate Commerce Law).
2. PL 89-97, the Federal Health Insurance for the Aged and its Code of Federal Regulations, Title 20, Chapter III, Part 405, "Conditions for Coverage of Services of Independent Laboratories" (Medicare Law).

Briefly the Clinical Laboratories Improvement Act of 1967, enacted by the 90th Congress, Section 5 of Public Law 90-174 amends Part F of Title III of the Public Health Service Act to include clinical laboratories under the licensing authority, which previously had extended only to biological products.

The amendment defines a clinical laboratory, interstate commerce, and general scope of licensing procedure, with provisions for revocation and redress, exceptions and exemptions, violations and the setting of penalties. Whatever standards for laboratory operation are devised by the Secretary, or his agency, must include: a) a quality control program, and b) participation in a proficiency testing program established by the Department of Health, Education and Welfare, in addition to c) the professional requirements of the personnel and d) the physical requirements of the laboratory operation.

The Medicare regulations similarly set up all inclusive standards of practice that independent and hospital laboratories must meet in order to comply with the Medicare law so that they are eligible for reimbursement for laboratory services.

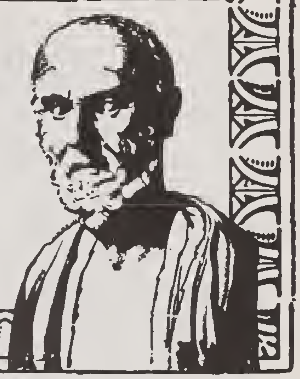
Suffice it to say that all legislation is the result of congressional hearings of public witnesses concerning proposals drawn up by either congress or the executive branch. The legislative bill is then reported to congress for debate and finally subjected to vote. If acted upon favorably, by congress, then it is submitted to the President for approval which he indicates by signature and date. The House and Senate are informed when the enrolled bill is delivered to the General Service Administration who in turn designates it as a public law, and numbers it for accession to the Archives. Codes of regulations are created by the responsible executive authority.

In the case of the CLIA and Medicare laws, these regulations were formulated by the executive branch, HEW, under broad responsibilities delegated by congress. Hence, Federal Regulations are not subject to majority vote of any deliberative body. Usually the regulations are drafted after consultation with public and private sectors of experts acting as advisory committees. Divergent opinions and contrary arguments are not necessarily invited until the regulations are drafted and published in the Federal Registry. The regulations usually take effect within 30 days of publication in the Federal Registry. Opposition to any or all of the proposed regulations follows prescribed due process procedures of a hearing and appeal.

The influence of these laws, their regulations, and any revisions will expand. State government agencies and professional associations are emphasizing proficiency testing to establish and maintain quality performance. Currently, physician office laboratories are feeling the effect of a new California State Law which requires "satisfactory performance in a proficiency testing program approved by the State Board of Health in laboratory procedures which such a laboratory performs". Recently, the Center for Disease Control of HEW proposed a revision in the Title 20 regulations which would bring under jurisdiction any physician's office laboratory operated by a group of at least three physicians. That revision has not been published as yet. Medicare regulations do not yet cover this area, but considerable interest is present because of the large amounts of reimbursement for laboratory tests paid to physicians who operate office laboratories.

It is difficult to agree that this will not benefit the patient, it will. But will it be equally just, fair and reasonable for the physician? The medical profession faces federalization of the entire health care delivery system unless adequate self regulations are voluntarily established and implemented. Nonetheless, we will be unable to avoid some degree of federal control. It is apparent to me that there is a real need to know how the legal process operates, and how our own rights can be protected through due process from adverse administrative actions.

William J. Jaffurs, M.D.



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but sometimes also of
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Hippocrates, *Precepts*, Chapt. I

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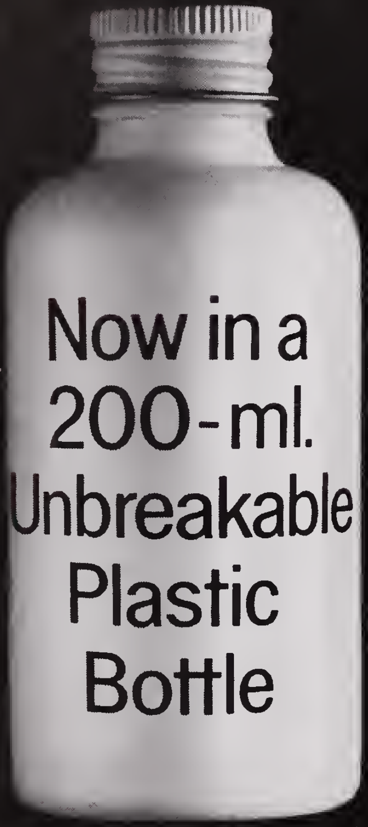
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The Umbilical Cord and Umbilical Artery Aplasia

JAMES C. PICKENS, M.D.*, WILLIAM J. JAFFURS, M.D.†, AND ATTA MEHRTASH, M.D.‡

The importance of the umbilical cord and its possible harmful significance in infancy may not be fully appreciated by the attending physician.[1-5] As a matter of fact, even now the cord is rarely considered except in those calamitous situations such as prolapsed cord, fetal distress, or compression of the cord. To many physicians the umbilical cord remains only a convenient handle to extract the placenta after the birth of the baby. This report details the startling significance of the absence of the umbilical artery, an observation that can be made easily.

In our study at the Columbia Hospital for Women we have determined the incidence of the single-artery cord, and the associated incidence of congenital anomalies in babies thus affected at birth and later at 1 to 5 years of age. Our purpose is to show the importance of examination of the cord at birth. Certainly, every diagnostic aid should be utilized to find hidden anomalies as early as possible so that corrective measures can be taken.

Historic Review

Probably the earliest recorded case of a single-artery cord can be attributed to Bauhin[6] in 1621. In 1870 Hyrtl[7] published the first important study of the blood vessels of the placenta and also described 14 cases of a single umbilical artery, in addition to 16 cases from the literature. In his series anencephaly was the most common malformation encountered. A single artery was reported in autosomal trisomy by Lenoske[8] in 1962, by Lewis[9] in 1962, and by Miller[10] in 1963.

In Froehlich's[11] series of 26,539 single births the incidence of a single umbilical artery on examination of the placentas was 0.76%. There were 20 stillborn infants; 6 died during the neonatal period and 3 died during infancy. Of the 174 infants who survived, 7 had lethal malformations. The incidence of congenital malformations in the remaining infants was 10.1% in those followed for 1 year.

Fujikura[12] collected cases from 5,972 consecutive deliveries. He found 38 cases of a single umbilical artery, and 7 of these had congenital malformations (18.4%). Sixteen of the remaining 31 infants were followed, and 3 of these were considered to have abnormalities in motor development. Other studies have reported similar findings, with the average incidence of a single umbilical artery about 1%.

Embryology and Anatomy

The umbilical cord is first recognized as such at about day 20–30. It is during this time that the body stalk becomes constricted into a cylindrical tube from which the cord will be formed. By the 8th week the cord has become a definite entity. The umbilical cord is formed by the expanding amnion which applies itself closely around the body stalk and yolk sac and crowds it into a tube-like structure. Hence, it is an auxiliary organ and not an outgrowth or extension of the body wall. A small portion of the extra-embryonic coelom is enclosed within the cord for a short time. From the 7th to the 10th week this portion of the extra-embryonic coelom contains coils of intestine, herniating into the cord. At 6 weeks the cord contains several distinct entities: the yolk sac, a pair of vitelline blood vessels to the yolk sac, the allantois and the allantoic umbilical blood vessels.

The vitelline arteries to the yolk sac subsequently become the blood supply for a portion of the intestinal tract. The umbilical arteries are formed in very young embryos from the 4th set of ventral branches and are those arteries accompanying the allantois that continue through the body stalk into the chorion, or primitive placenta. These arteries remain paired, and by the end of the 4th week each artery acquires a secondary connection with a dorsal intersegmental branch of the aorta, and the ventral stem disappears. This dorsal stem becomes the external iliac and hypogastric arteries connecting directly to the placental circulation.

The right and left umbilical veins are about equal in size in the 4.5 mm embryo, both draining into the sinus venosus. However, even in the 5 mm embryo the left umbilical vein is much more prominent, and in the 9 mm embryo the right umbilical vein has disappeared completely, the left umbilical vein draining into the hepatic sinusoids and later into the ductus venosus. From this time, the umbilical cord is essentially unchanged, except to grow in size and length, its length approximating the length of the fetus at each stage of development.[13]

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The umbilical cord normally contains 3 vessels and Wharton jelly. This jelly probably serves to make the cord less susceptible to twists and compression. The 2 arteries have a thick, muscular coat and are capable of strong contractions. The ability to narrow the lumen to a slit-like orifice probably is due to the double spiral course of the muscular fibers. Nerve-like structures have recently been identified in the cord by the electron microscope, but as yet their exact nature has not been determined.

The spiral twist of the cord has been the subject of some discussion. Edmonds[14] proposed the theory that the direction of the twist of the normal umbilical cord is determined by the position of the embryo in relation to the effective axis of tortion of the early gravid uterus. According to his theory, the twist of the normal umbilical cord is the result of a rotatory movement imparted to the embryo by the asymmetrical contraction of the uterus. The normal direction of twist is from right to left, probably because the asymmetric contractions usually begin on the right side and sweep to the left.

The length of the cord usually approximates the length of the fetus at any stage of development. It must be long enough to reach from the attached placental site to the level of the vulva, usually a distance of 32 cm in a fundally located placenta. The average length is about 55 cm but has been reported up to 181 cm. The cord may be inserted at any place or may even end on the membranes, as in velamentous insertion.

Materials and Methods

A segment of the umbilical cord approximately 2-3 cm long was cut from the fetal end of the cord at least 10 cm from the fetus. These were preserved in isotonic saline until the next morning when they were examined grossly by a resident physician. It should be noted that all the cord specimens were examined by either 1 of 2 obstetricians so that there would be uniformity to the study. Cords that were thought to be abnormal were sent to the laboratory and a gross and microscopic examination performed by the pathologist. An entry was made on a questionnaire and the mother later questioned as to:

1. Maternal past history
2. Family history
3. History of previous pregnancies
4. History of the present pregnancy
5. Siblings with congenital anomalies
6. Exposure to drugs, X-rays.

The infant was then examined by the obstetrician, pediatrician, and/or resident physician. All were notified about the abnormal number of vessels in the cord. Stillbirths and neonatal deaths were autopsied whenever possible.

For the 1 year follow-up a letter was sent to the obstetrician and the pediatrician reminding them of the abnormal umbilical cord findings and asking them for a follow-up on the infant in respect to congenital anomalies, growth and development, and any recurrent problem. This was repeated at the end of 5 years.

Results and Discussion

This study series represents 10,223 consecutive deliveries at the Columbia Hospital during the period December 1, 1963 through December 31, 1965.

Seventy-five cases were collected that had only one artery and one vein. Three of the 75 examined on microscopic section contained 1 vein, 1 artery, and a very hypoplastic artery that could carry very little, if any, blood. There were 2 cases in which there was an extra number of vessels; 1 contained 3 arteries and 2 veins, and the other contained 2 arteries and 2 veins. Also, there was 1 in which there were actually 2 cords, connected together by a transparent membrane. These 3 cases are not included in the final totals.

Maternal factors. The ages of the mothers ranged from 14 to 43, the average being 26.5 years. As can be seen from table 1, the majority fall in the 20 to 35 age-group. Almost all of the primigravidas were in the age group below 30, but as shown in table 3, the patients with abnormal babies showed a more even distribution, with high percentages both above and below 30 years of age. Other reports have shown a higher incidence of abnormal babies in the very young and very old mothers, but our study does not bear this out. In our series we could show no particular predisposition in any age group for a certain disorder such as anencephaly. The age distribution is compatible with a series of controls from the same hospital.

Table 2 shows the number of abnormal babies compared with the gravidity of the mothers. There is a fairly even distribution up to gravida 5 where the percentage decreases. In a larger series of cases the similarity would perhaps be more striking since the gravidity roughly corresponds to the maternal age. In the 18 cases that were abnormal, the 18 women had in the past produced a total of 22 normal infants out of a total of 23 pregnancies. Only 1 woman gave a history of previous abortion at an early stage of pregnancy, and it is not known whether or not the fetus was abnormal.

The other 57 women had a total of 145 pregnancies with 125 liveborn

TABLE 1: Ages of Mothers and Gravida

Mother's Age in Years	Primi-gravida	Multi-gravida	Total
Below 20	8	2	10
20-25	15	15	30
26-30	4	9	13
31-35	0	14	14
36-40	2	5	7
Above 40	0	1	1

TABLE 2: Gravidity

Gravida	Number	Abnormal Babies	%
1	30	7	23
2	18	4	22
3	11	4	36
4	6	1 ?*	17
5	3	1	33
6	1	0	0
7	4	1	25
8	1	0	0
9	1	0	0

* Baby died with meningitis at age 4 months, also had questionable brain damage.

children, who are living and well. Fourteen of the abortions occurred in 3 women. This abortion rate in the group does not bear out the poor prognosis reported by some investigators. This abortion rate of 14% compares favorably with a control series. Certainly, there is no evidence of a decreased fertility in these women.

Much has been written about the influence of maternal diseases in relation to abnormal umbilical vessels. Benirschke and Brown[15] reported a high incidence of pre-eclampsia (30%). This has not been confirmed by other investigators. In table 4 the diseases recorded in the total of 75 patients are tabulated. Out of 75 patients there were 7 patients with pre-eclampsia, defined as a BP of 140/90 or greater. This represents an incidence of 9.3%. However, in these 7 patients 3 had abnormal babies for an incidence of 43%. It would, therefore, seem that if the mother is pre-eclamptic and has a baby with an abnormal number of vessels in the umbilical cord, this baby should be watched closely during the first few hours of life for hidden anomalies and perhaps further studies performed.

TABLE 3: Abnormal Babies in Relation to Maternal Age

Mother's Age in Years	Number	Abnormal Babies	%
Below 20	10	1	10
20-25	30	9	30
26-30	13	1	8
31-35	14	4	29
36-40	7	2	29
Above 40	1	1	100

TABLE 4: Maternal Complications

Complication	Number	Abnormal Babies	%
Pre-eclampsia (BP 140/90 plus)	7	3	43
German measles, first trimester	4	3	75
Polyhydramnios	3	3	100

Especially noteworthy is the high percentage of abnormal babies in the mothers who had German measles in the first trimester. Of these 3 babies, 2 were diagnosed as normal by the pediatrician at birth and later were found to have cardiac anomalies as well as cataracts.

In review of the histories of the 75 women who delivered an infant with a single umbilical artery and especially of the histories of the 18 women who had abnormal babies, there was found no evidence of any factor that might affect the general health. We found no evidence of poor nutrition or any chronic debilitating disease. There was only 1 case with a history of erythroblastosis fetalis, but the infant was now normal.

There were 3 cases in which polyhydramnios was diagnosed. In all of these the baby was grossly abnormal. All fetuses were diagnosed prepartum as having a fetal anomaly, and 1 was terminated by saline injection. There was no maternal history of previous hydramnios.

There was no mother who had had a baby with a congenital absence of an umbilical artery. There have been reports in the literature of siblings being born with the defect[16], but there is no evidence that this occurs any oftener than by chance. There were several mothers in our series who had had a previous infant with a

TABLE 5: Fetal Abnormalities

Musculoskeletal:	
Multiple skeletal deformities	4
Tibial torsions	1
Marked clubbing of both feet	1
Gastrointestinal:	
Imperforate anus	3
Inguinal hernia	4
Diaphragmatic hernia	1
T-E fistula	1
Genitourinary:	
Labial fusion	1
Absent vagina	1
Multicystic kidneys	3
Undescended testicle	2
Hypospadius	2
Extrophy of bladder	1
Cardiovascular:	
Patent ductus arteriosus	1
Tetrology of Fallot	3
Undiagnosed heart disease	1
Idiopathic supraventricular tachycardia	1
Neurological:	
Hydrocephaly	2
Anencephaly	3
Cataract	3

TABLE 6: Intrauterine and Neonatal Deaths

Case No.	Birth Weight	Sex	Autopsy Findings
1.	1 lb.	M	Anencephaly; Tetrology of Fallot; multiple skeletal anomalies; stillborn
2.	3 lb.	F	Anencephaly; multiple skeletal anomalies; omphalocele, polycystic kidneys; stillborn
3.	5 lb., 5 oz.	M	Neonatal death from meningitis; brain damage
4.	8 lb., 7 oz.	M	Hydrocephalic, T-E fistula; double colon; stillborn
5.	3 lb., 3 oz.	F	Anencephaly; multiple skeletal anomalies; stillborn
6.	3 lb.	M	Multiple skeletal anomalies; congenital heart disease; multicystic kidneys; stillborn
7.	5 lb.	M	Hydrocephalic; hypospadius; undescended testicle; imperforate anus; stillborn

congenital defect, but all of these were relatively minor defects such as clubfoot, extra digits, etc. In our series only major, life-threatening defects were recorded. Extra digits, birthmarks, hemangiomas, etc. were not considered. There was no higher incidence of prepartum, intrapartum, or postpartum complications in the hospital.

Two babies in our series were delivered by caesarean section. Both of the mothers had a previous caesarean and both babies were normal. There was no demonstrable increase in obstetric complications, and there was actually a slight decrease in the use of forceps. One infant required trocar drainage of the fetal head because of hydrocephaly, but the delivery 2 hours later was uneventful.

There was no increase in pre- or post-partum infection in the 75 mothers studied. The percentage of patients with premature rupture of the membranes was the same as in the control group.

Placental factors. An increase in the number of abnormal placentas has been reported by several authors.[17] This was not the case in our series. There was the usual number of placental infarcts, dilated vessels, etc., but there was no increase in abnormal placentas from the 75 cases studied over the number normally expected in routine deliveries. There was no case of amnionitis or placentitis.

Infant factors. There were 10,223 deliveries at the Columbia Hospital for Women during the period of study. 75 infants were found to have a single umbilical artery out of a total of 10,319 infants born, for an overall incidence of 0.73%. This figure is lower than the figure quoted in most series, but we believe it represents as accurate an estimation of the rate that can be expected. No selection of any kind was employed, and the cords were examined without prior knowledge of the maternal history.

During the period of study, 7,384 Caucasian babies were born. Of these, 48 babies were born with no abnormality other than absence of 1 umbilical artery. However, 16 Caucasian babies were born with absence of 1 umbilical artery and major congenital defects. The incidence, therefore, of congenital absence of an umbilical artery is 0.87%, which is much higher than the total of the group.

Conversely, a total of 2,935 nonwhite babies were born during the period in question. Nine of these infants had no abnormality other than a single umbilical artery, and 2 had major malformations. The incidence in this group was 0.37%, a much lower figure than for the Caucasian babies.

The percentage of abnormalities in infants discovered to have a single umbilical artery also shows a marked difference when race is considered. Out of a total of 64 Caucasian babies, there were 16, almost 30%, with malformations. In the non-white babies, there were 11 with 1 umbilical artery and only 2 were abnormal, a percentage of 18%. We are unable to explain this marked difference in the races.

There were 96 sets of twins born during this time. Several series have mentioned the increase in the incidence of a single-artery cord, quoting figures as high as 7% of all twins. In our series there was not a single case of a twin born with 1 umbilical artery. We are at a loss to explain this finding since the twin cords were examined in exactly the same way as the other cords.

Prematurity might be expected to play some role in any series dealing with congenital defects. If we consider any infant premature who weighs 5 lbs. or less, out of the 75 infants studied there were 11 who meet this criteria, a percentage of almost 15%. This is compared with a prematurity rate of 8.8% for all babies born in the hospital and shows a considerable increase in prematurity for infants with a single umbilical artery.

An even more striking increase can be shown if we consider only the 18 cases in which the infant was considered abnormal. In this group there were 7 babies, 38.8%, with birth weights of 5 lbs. or less. However, even though there was a high percentage of prematures in this group, not 1 of the deaths that occurred could be attributed to prematurity. All infants who died did so as a result of their anomalies.

Several reports in the literature have emphasized the predominance of males in the infants with a single umbilical artery. In the present series of 75 infants there were 18 who had congenital defects. Of the 18 infants, 10 (55%) were males. In the remaining 57 infants there were 25 (44%) males. These percentages are not significantly different from the expected ratio that might occur by chance.

One year follow-up. The letter and questionnaire sent to the pediatrician and obstetrician 1 year after the birth requested the results of check-ups, laboratory tests, and X-rays, or electrocardiograms. We also inquired if any abnormal findings had resulted.

It was difficult to follow the staff cases, since we do not have a pediatric clinic at this hospital, and also many of the patients delivered are from the House of Mercy, a home for un-wed girls; most of the infants are put up for adoption.

Our inquiries resulted in a total of 43 replies, 55.1%, in which the outcome is known. This includes 7 infants who died and were autopsied. Of the 75 infants having a single artery in the umbilical cord, 12 (16%) were diagnosed as having congenital anomalies at birth. As a result of the follow-up questionnaire, we discovered 6 more babies with congenital anomalies who were thought to be normal at birth. Thus, in our series, we found that 24% of the babies born with a single umbilical artery will have congenital anomalies, but 1/3d of these anomalies will not be discovered at birth and can only be diagnosed as the baby is followed. In 2 of the 6 babies who were later discovered to have congenital anomalies there was a history of German measles in the first trimester. Therefore, it is imperative that any baby whose mother was exposed to German measles in the first trimester be followed with extreme care, and further studies, as will be outlined below, should be performed.

Five-year follow-up. A similar letter and questionnaire were sent to the pediatrician and obstetrician 5 years after birth. Again, we had the same type of results. More of the patients had been lost to the study, and fewer replies were received. One additional infant was discovered to have congenital heart disease during the 2nd year of life. Corrective surgery had been performed successfully. This raised the percentage of babies born with a single umbilical artery who manifested congenital anomalies to 25%.

Conclusions and Recommendations

The presence of a single umbilical artery in an infant is an important finding and should alert the obstetrician and pediatrician to observe for other anomalies. The examination is simple and takes very little time and effort to perform. As shown in our series, the incidence is approximately 0.73% of all infants: 0.87% of white infants and 0.37% of non-white infants. The reason for this difference is not understood. In this hospital this incidence means that approximately 3 infants per month will be born with only 1 umbilical artery, and of these, almost 1/3d will have a congenital defect. Even more important is the fact that, of the infants with a congenital defect, 1/3d are unrecognized at birth.

The most important single factor in the maternal history seems to be viral diseases in the first trimester. Every indicator of hidden congenital defects at our disposal should be used to diagnose these disorders before they manifest themselves in overt disease.

In all infants born with a single umbilical artery a diagnostic work-up should include the following:

1. **Medical history.** This should include maternal history concerning chronic debilitating diseases, drug intake, and of X-ray films taken during the first trimester. Previous pregnancies should be reviewed as to the presence of abortions that may have been examined in other hospitals, with special reference to cytogenetic studies. A review of the present pregnancy should include inquiries as to viral diseases, especially German measles. A history of the disease in childhood does not rule out the possibility of the patient having the disease in this pregnancy, since many diseases are misdiagnosed as German measles. This was the case in 2 or our patients. The prenatal course should be reviewed, since an increased incidence with toxemia has been reported, although our experience does not bear this out. Our series does show that if the mother has a history of toxemia the infant with 1 umbilical artery is more likely to have a congenital defect.

2. **Physical examination.** The obstetrician who delivers an infant with congenital absence of an umbilical artery is in the unique position to examine the infant while suction of the throat, stomach, etc. is being performed. He has the opportunity to observe the infant during the first breath and during the first few minutes of life, probably the most critical in the infant's life. An observation then may save the life of the infant, for example, in 1 with a tracheoesophageal fistula. Even if the infant appears normal the finding of a single umbilical artery should be noted on the chart and confirmed microscopically. The pediatrician should be notified of this finding so that the infant can be observed and examined with special care. Special attention should be given to the genitourinary and cardiovascular systems, for it is there that hidden anomalies will most likely be found. The parents should be informed of this finding and its possible implications. Even if the infant appears normal, checkups are of ut-

most importance, and any abnormal behavior should be reported to the physician.

The finding requires close cooperation of the parents and the physician. As we have shown in our series, an additional 1/3d of the anomalies were discovered during the first year of development, and possibly even more would have been discovered if we would have been able to obtain a better follow-up. We believe that most of the anomalies will be picked up by the end of the first year. Any abnormalities discovered by the physician should lead to further testing.

3. **Cytogenetics.** This is becoming more readily available. It is an expensive but rewarding test in the diagnosis of the "suspect" infant.

4. **Intravenous urogram.** In 1 series of infants with a single umbilical artery 30% of the cases were found to have renal abnormalities.[18] Bagdassarian[19] considers a single umbilical artery an indication for intravenous urography. This can be performed through the umbilical vein. We do not believe this should be done routinely but reserved for the infants with the following conditions:

a. A strong family history of congenital anomalies
b. An infant with other congenital anomalies
c. An infant with a genitourinary anomaly (hypospadius, etc.) or with a history of urinary-tract infection.
d. Any infant of a mother with hydramnios
e. History of pre-eclampsia in the mother.

5. **Electrocardiogram.** This should be done routinely in any infant with a single umbilical artery, especially since the evaluation of the heart of a newborn infant is difficult even in the best of hands.

6. **Chest X-ray films.** This is another test which can be performed easily and may give a clue as to the cardiac status. Also, congenital defects in the lungs or diaphragm may be diagnosed.

7. **Esophagogram.** This should certainly not be routine but is very useful in the infant suspect of esophageal atresia, or T-E fistula.

8. **Sonogram.** This is especially useful for intra-abdominal masses and should be used prior to intravenous urogram.

Summary

A discussion of the umbilical cord, its development and anomalies, has been presented with special reference to congenital absence of an umbilical artery. Seventy-five infants were discovered to have an absence of 1 umbilical artery from a total of 10,223 consecutive births (10,319 infants) for an overall incidence of 0.73%. Of these, 16% were diagnosed as having congenital anomalies at birth, an additional 8% was discovered by the end of the first year, and still another 1% by the end of the second year, to give an overall incidence of 25% by the end of infancy. The clinical findings in the 75 infants with this disorder have been reviewed. Recommendations concerning diagnostic studies and follow-up examinations have been enumerated. It is hoped that more concern for the simple examination of the umbilical cord will be stimulated, resulting in earlier and better treatment of congenital diseases in the newborn.

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Early Obstetric Diagnosis by Diagnostic Ultrasound*

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Ultrasound is sound waves above the limit of human hearing, that is, in excess of 20,000 cycles/second. The frequency range in diagnostic ultrasound is 1-10 million cycles/second.

The sound is transmitted and received by a transducer mounted in a probe. Reflection of echoes takes place at interfaces between tissues of different densities. The reflected sound is converted in electrical impulses which are displayed on an oscilloscope screen giving a 2 dimensional representation of the area scanned. A permanent record is then taken from the screen by a Polaroid camera.

The energy produced in diagnostic ultrasound is of low value in the range 1-10 milliwatts/square cm. Thus far no toxic effects have been demonstrated to either the fetus or the mother.[1]

No preparation of the patient is required other than partial filling of the bladder, which displaces small bowel from the pelvis. The patient is examined in a supine position, and after the abdomen is smeared liberally with mineral oil multiple scans in the saggital and transverse planes are performed. There is no discomfort to the patient, and the entire procedure takes 20-30 minutes to complete.

Since obstetrical cases are presented in which B-mode ultrasound scanning was in making a diagnosis prior to the twentieth week of pregnancy.

From as early as 4 weeks gestational age the developing amniotic sac may be visualized (Fig. 1). This developing sac may be demonstrated as a separate structure until about 12 weeks when there is fusion of the decidua vera and decidua capsularis.

By demonstrating 2 or more amniotic sacs it is possible to make the diagnosis of multiple pregnancy in the first trimester. Needless to say, monozygotic multiple pregnancies will not be demonstrated by this method.

Illustrative Cases

Case 1. L. G., a 27-year-old G1, PO, prior to conception had received Perganol for ovarian stimulation. Gonadotrophin levels after conception were found to be very high, and molar or multiple pregnancy were considered. Ultrasound examination at 8 weeks menstrual age demonstrated 3 separate amniotic sacs within the uterine cavity (Fig. 2). At 27 weeks 3 fetal skulls were outlined (Fig. 3). A plain X-ray film of the abdomen confirmed the presence of triplet pregnancy.

Anencephaly. The incidence of this condition has marked geographic variation ranging from 0.12-3.50/1000

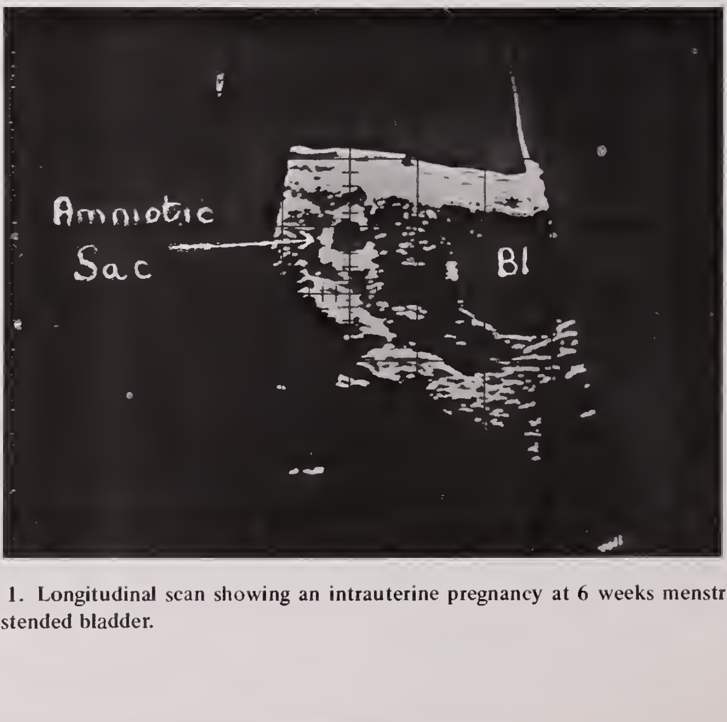

FIG. 1. Longitudinal scan showing an intrauterine pregnancy at 6 weeks menstrual age. Note distended bladder.

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births.[2] No genetic defect has been described, although there is a tendency to recurrence (see case report no. 3). The defect is characterised by the absence of the occipital and parietal bones. Until the introduction of diagnostic ultrasound the only definite method of diagnosis was by radiological studies of the fetal skeleton.[3] Most radiologists, however, would be unwilling to make a diagnosis prior to 20 weeks.

With modern ultrasound equipment it is possible to demonstrate the fetal skull as early as 12 weeks and consistently from 16 weeks to term. If the fetal skull cannot be demonstrated after 2 separate examinations, anencephaly is highly probable. It is advisable, however, before making the diagnosis to perform a pelvic examination to exclude a deeply engaged fetal head.

Case 2. R. M., age 35, G4, P3. This patient had previously delivered 3 infants with multiple fetal anomalies. In the present pregnancy she was examined by ultrasound early in the first trimester, and an apparently normal intra-uterine pregnancy was found. She was then re-examined at 2-week intervals. By 18 weeks no fetal skull could be outlined, although the fetal trunk and limbs were noted (Fig. 4). A diagnosis of anencephaly was made at this point and confirmed by X-ray film at 26 weeks. The patient was subsequently delivered of an anencephalic infant.

Case 3. A. L., a 29-year-old G5, P3, Abl., had had 3 anencephalic babies after 20 weeks and a spontaneous abortion before 20 weeks. The aborted fetus showed multiple central nervous-system abnormalties. The patient was first examined by ultrasound at 16 weeks, when a well-formed fetal skull measuring 3 cm was visualized (Fig. 5). Examination at 18 weeks again revealed a well outlined skull with no evidence of skull anomaly. The patient was reassured.

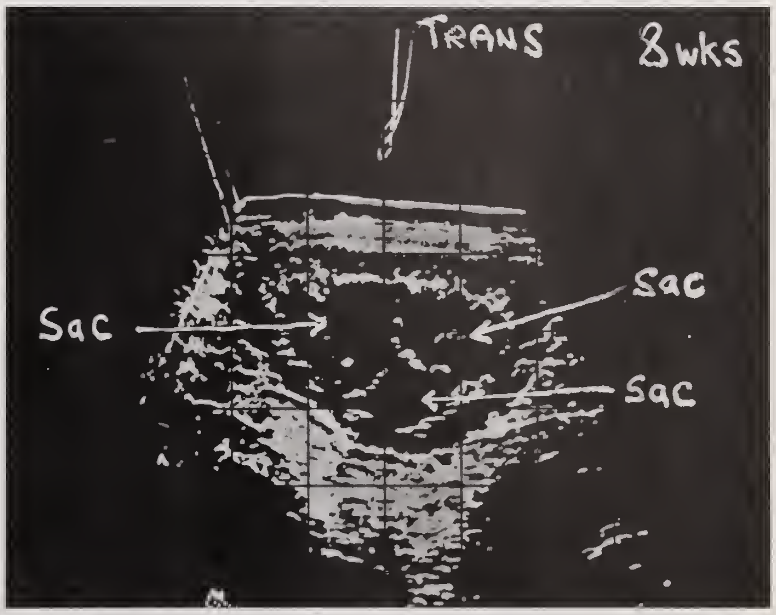

FIG. 2. Transverse scan of uterus at 8 weeks menstrual age showing 3 amniotic sacs.

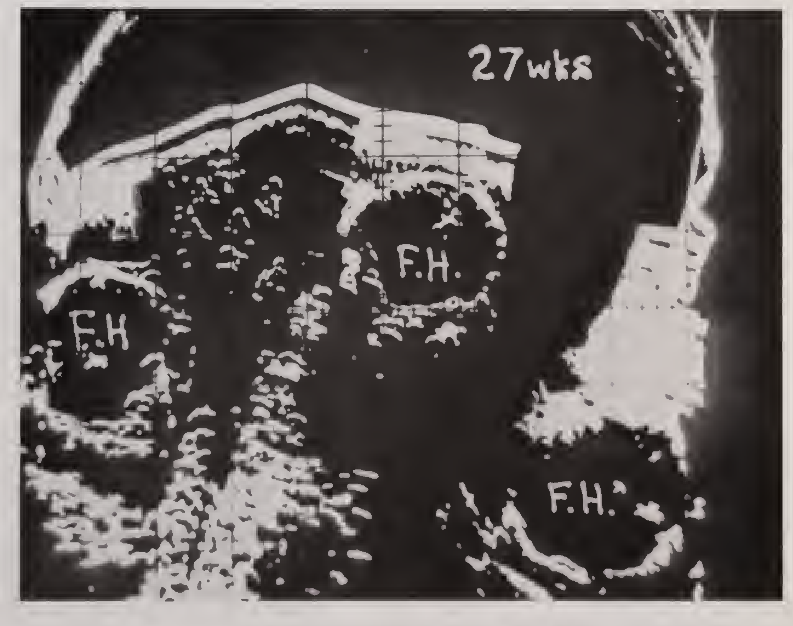

FIG. 3. Composite scan of uterus displaying 3 fetal skulls at 27 weeks.

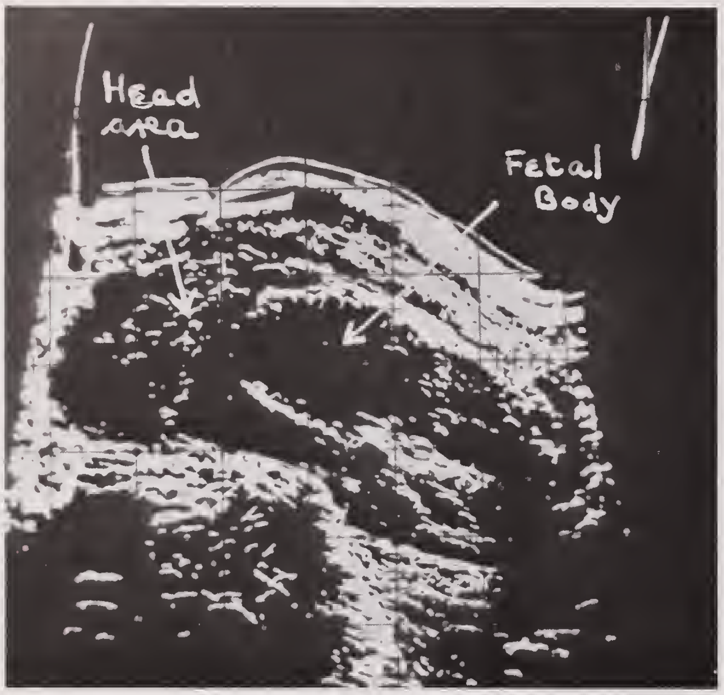

FIG. 4. Longitudinal scan of 18-weeks pregnancy. Breech presentations. No fetal skull visualized.

At 22 weeks an X-ray film was taken, and although fetal spine and small parts were seen, no skull shadow was described (Fig. 6). The patient was re-examined at 24 weeks by X-ray, when a normal-appearing skull was noted (Fig. 7). She later delivered a normal infant with no evidence of fetal skull anomaly.

Discussion

The use of diagnostic ultrasound in obstetrics and gynecology is now well established,[4] and at Columbia Hospital it is used routinely for the diagnosis of early pregnancy, placental localization, estimation of fetal maturity, and confirmation of suspected pelvic tumors. It is particularly useful in the first 20 weeks of pregnancy when traditional radiological methods are either contraindicated or of little value.

Anencephaly is a not uncommon fetal anomaly, and most authorities would recommend a therapeutic abortion of such pregnancies if diagnosed early enough. Although X-ray will make a positive diagnosis, this is often after the legal limit set by most communities for this procedure. Ultrasound offers another method of diagnosis at an earlier stage of pregnancy. In our case of repeated anencephalic deliveries, reassurance of nonrecurrence was sought and given by ultrasound at 16 weeks. This was 8 weeks earlier than a similar reassurance could be given by X-ray examination.

The increased incidence of multiple pregnancies following ovarian stimulation is well known.[5] Early diagnosis could conceivably reduce the increased fetal loss associated with such pregnancies.[6] Routine scanning of such cases would reassure the doctor and allay the fears of the patient.

The cases presented are only 3 of over 1,200 patients examined by ultrasound at Columbia Hospital since its introduction about 1 year ago. The majority of these were obstetric

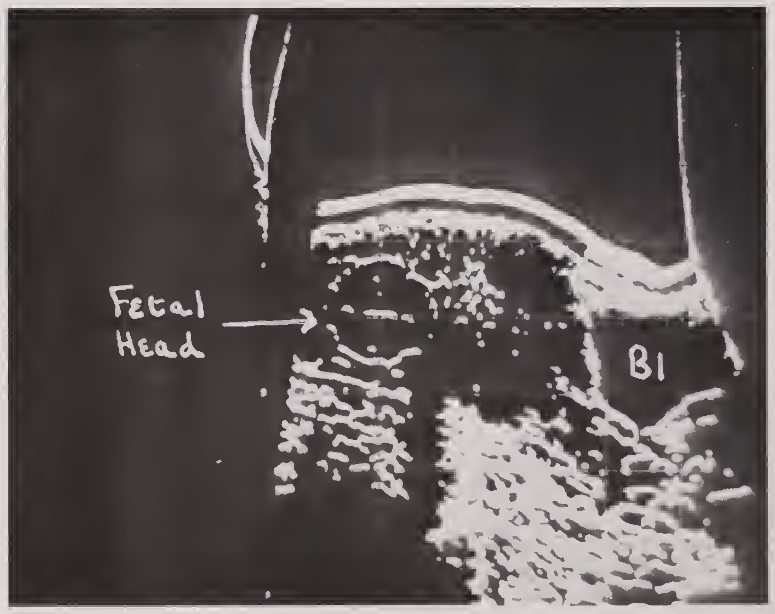

FIG. 5. Longitudinal scan of 16-weeks pregnancy showing fetal skull. Note midline echoes from the falx and distended bladder.

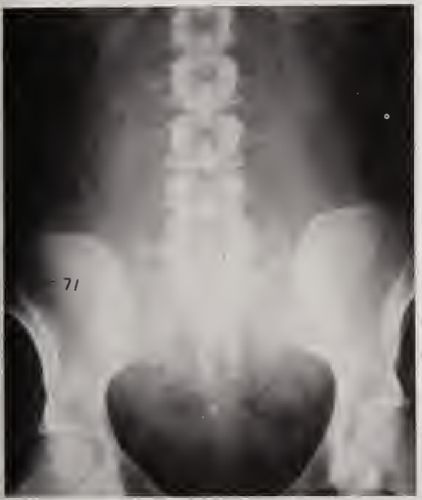

FIG. 6. Film at 22 weeks. No skull visualized although spine and small parts are seen.

patients, many of whom were in early pregnancy. A large number of first trimester bleeders were examined to rule out ectopic pregnancy and molar pregnancy, and to monitor amniotic sac development. It was found that a well outlined amniotic sac meant well for the pregnancy in the majority of cases. The management of missed abortion, which at times is most difficult, was made much easier by ultrasound scanning when laboratory tests were ambivalent.

The menstrual history of patients requesting therapeutic abortions is notoriously unreliable. After 14 weeks the measurement of the biparietal diameter is an accurate method of dating the pregnancy. Our upper limit for therapeutic abortion has been a reading of 5.0 cm which we have found to be consistent with a gestational age of 20 weeks.

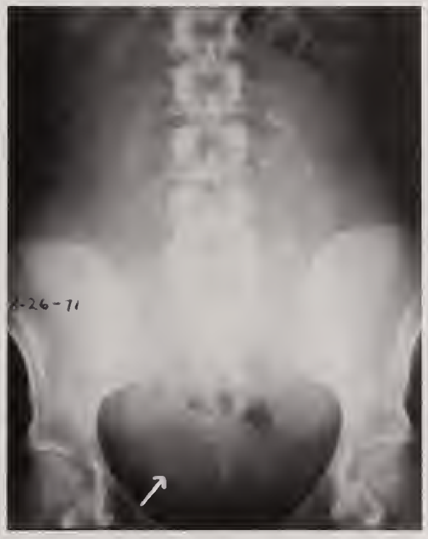

FIG. 7. Film at 24 weeks. Normal fetal skull outlined in pelvis.

At present most of our observations on early pregnancy are of a gross nature, e.g., amniotic sac, fetal head, placenta. It is hoped that with improved equipment giving greater resolution more detail of early fetal development may be visualized. It is not far-fetched to surmise that in the near future early ultrasound examination will become as routine in the management of the pregnant patient as serology and blood typing are today.

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An Evaluation of Newborn Mortality and Prematurity Trends at Columbia Hospital for Women, Washington, D.C. 1967–1970

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In July 1968 intensive care treatment for the newborn began at Columbia Hospital for Women (CHW). On January 1, 1969 the Intensive Care Unit for newborn infants was officially opened. Since then the quality of care at CHW has steadily increased. This is shown by a decreasing neonatal mortality rate.

Before the Intensive Care Unit started the sick infants were transfered to other hospitals in the area better equipped for treating newborn infants. In 1967, 62 infants were transfered, whereas in 1970 the number had fallen to 28 cases, all congenital anomalies potentially in need of surgical correction. Columbia Hospital for Women has no facilities for infant surgery.

Mortality

Table 1 shows deaths occurring before discharge arrayed by birthweight group, calendar year, and staff versus private population.

Table 2 shows all livebirths similarly arrayed.

Table 3 shows our weight-specific neonatal death rates. These rates include deaths prior to discharge and deaths among infants transfered (if within 28 days of life). Newborns who weighed 500 gm or less are not included. Total death rates show that 1970 is clearly the best year for both staff and private babies if birth weight is not considered. For both staff and private populations the total death rates decreased substantially between 1969 and 1970. If we shift our attention, however, to babies weighing 501-2500 gm, we see a difference in the mortality trend between the staff

TABLE 1: Deaths 1967-1970

Weight Group	Total Hospital				Staff				Private			
	1967	1968	1969	1970	1967	1968	1969	1970	1967	1968	1969	1970
501-1000 gm	16	20	26	18	3	3	3	3	13	17	23	15
1001-1500 gm	11	18	22	8	0	6	7	4	11	12	15	4
1501-2000 gm	7	10	7	6	3	1	0	1	4	9	7	5
2001-2500 gm	12	6	4	9	2	0	1	2	10	6	3	7
above 2500 gm	15	14	11	9	4	3	3	1	11	11	8	8
Total	61	68	70	50	12	13	14	11	49	55	56	39

TABLE 2: Liveborn 1967-1970

Weight Group	Total Hospital				Staff				Private			
	1967	1968	1969	1970	1967	1968	1969	1970	1967	1968	1969	1970
501-1000 gm	19	22	29	21	3	3	5	3	16	19	24	18
1001-1500 gm	28	33	47	23	5	12	15	4	23	21	32	19
1501-2000 gm	83	74	59	83	27	22	18	11	56	52	41	72
2001-2500 gm	245	228	251	289	67	65	57	62	178	163	194	227
above 2500 gm	4306	4043	3969	4187	823	792	733	749	3483	3251	3236	3438
Total	4681	4400	4355	4603	925	894	828	829	3756	3506	3527	3774

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TABLE 3: Weight-specific Death Rates/1000 Liveborn. 1967-1970.

Weight Group	Total Hospital				Staff				Private			
	1967	1968	1969	1970	1967	1968	1969	1970	1967	1968	1969	1970
501-1000 gm	842.1	909.0	897.0	857.1	1000	1000	600	1000	812.5	895.0	875.0	833.3
1001-1500 gm	392.9	545.5	468.0	347.8	0	500	466.7	1000	478.3	571.4	531.3	210.5
1501-2000 gm	84.3	135.1	67.8	72.3	111.1	45.5	0	90.9	71.4	173.1	170.7	69.4
2001-2500 gm	49.0	26.3	15.9	31.1	29.9	0	17.5	32.3	56.2	36.8	15.5	30.8
above 2500 gm	3.5	3.5	2.8	2.1	4.9	3.8	4.1	1.3	3.2	3.4	2.5	2.3
Total	13.0	15.5	16.1	10.9	12.9	14.5	16.9	13.3	13.0	15.7	15.9	10.3
Total 501-2500 gm	122.7	151.3	152.8	98.6	78.4	98.0	115.8	125.0	139.2	172.5	164.9	92.3

and private babies. Premature babies had a moderate increase in mortality over the same time period. This analysis suggests that we must try to improve our care for premature staff babies.

Table 4 is like table 3, except that race and sex categories have been added, and birth weight has been dichotomized at 2500 gm. Among our low-birth-weight infants, the staff babies, regardless of race, have lower death rates than their private counterparts for the total 4-year period. As seen in table 3, however, the staff death rates for 1970 exceed the private, and this is true for both races.

For the small babies the table shows higher male death rates for both races in both the staff and private populations. It should be noted, however, that for both private and staff, white baby male death rates are more than 2 times as great as female death rates. For blacks the male/female differential is not as great.

Table 4 also shows that for premature babies, white mortality rates are higher than black in both populations.

The calendar year trends observed in table 3 for premature babies apply equally to white and black babies within the staff and private categories.

In large babies the mortality patterns across these various classification dimensions are different. In contrast to small babies, staff total rates over the 4-year period for both races exceed the private rates. The small number of deaths in the above 2500 gm babies does not permit any further observations.

The addition of race and sex to our mortality data analysis permits us to conclude that staff mortality rates rose steadily over the 4-year period regardless of race, and private mortality rates declined steadily regardless of race.

The stillbirth rate for the hospital (about 10/1000 livebirths) which is somewhat less than the national average (15.6/1000 in 1967), has not changed materially over the 4-year period.

In spite of a declining mortality rate, the hospital prematurity rates have increased.

Prematurity

We define prematurity in terms of birthweight (501-2500 gm). Some idea of the importance of prematurity trends can be gained by looking at table 3. In 1970, for example, the mortality risk for babies above 2500 gm is 2.1/1000 livebirths whereas babies weighing 501-2500 gm have a mortality risk of 98.6/1000 livebirths. Thus, premature newborns have nearly 50 times greater chance of dying than full-weight newborns. For this reason it is important to analyze the hospital birth weight data.

Table 5 shows prematurity trends for the entire hospital population of newborns, as well as staff and private portions of the total. Overall, prematurity rates increased over the 4-year period. In 1967 the CHW prematurity rate was slightly below the national rate of 8.2%. National data are not yet available for the other years, but the national rate does not change much from year to year, but the current overall CHW prematurity rate of 9.04% probably exceeds the national average. When broken down into staff and private components, interesting countertrends become evident. Staff rates remained more or less constant over the 1967-1969 period but dropped in 1970. Private rates were

TABLE 4: Death Rates/1000 Liveborn, Divided into 501-2500 gram and above 2500 gram groups and categorized by pay status, race and sex

	501-2500 Grams					
Year	Staff White			Private White		
	Male	Female	Total	Male	Female	Total
1967	125.0	83.3	100	236.0	80.4	149.3
1968	176.5	0	125.0	278.5	75.9	177.2
1969	375.0	0	166.7	174.6	128.7	146.3
1970	0	250.0	166.7	166.7	63.3	112.6
Total	200.0	60.6	132.4	217.8	88.9	146.9
	Black			Black		
1967	138.9	21.7	73.0	96.8	100.0	98.6
1968	64.5	106.4	89.7	153.8	204.5	177.1
1969	176.5	22.7	89.7	258.6	136.4	193.5
1970	117.6	102.6	109.6	116.3	31.3	71.4
Total	125.9	62.5	90.0	158.6	101.6	129.0
	Above 2500 Grams					
	White			White		
	Male	Female	Total	Male	Female	Total
1967	8.0	0	3.9	2.0	3.8	2.8
1968	8.6	0	4.6	4.7	2.4	3.6
1969	0	12.3	5.9	2.4	2.6	2.5
1970	0	0	0	2.7	0.9	1.8
Total	2.5	2.6	3.8	2.9	2.5	2.7
	Black			Black		
1967	0	10.7	5.3	8.6	0	4.6
1968	6.6	0	3.5	2.7	0	1.4
1969	3.6	3.6	3.6	0	2.5	1.2
1970	3.5	0	1.7	7.8	0	4.1
Total	3.5	3.5	3.5	5.1	0.6	3.0

TABLE 5: Prematurity Rates/100 Livebirths (Prematurity = 501-2500 grams)
(All Hospital)

1967	1968	1969	1970
8.01%	8.11%	8.86%	9.04%

Staff

1967	1968	1969	1970
11.03%	11.4%	11.47%	9.65%

Private

1967	1968	1969	1970
7.27%	7.27%	8.25%	8.90%

TABLE 6: Prematurity Rates/100 Livebirths (Prematurity = 501–2500 grams)

Year	Staff White			Private White		
	Male	Female	Total	Male	Female	Total
1967	6.01	8.45	7.28	5.70	7.82	6.72
1968	12.78	6.47	9.96	5.81	5.96	5.89
1969	8.33	11.0	9.63	4.83	7.94	6.37
1970	2.63	5.19	3.92	6.01	6.90	6.43
Total	7.98	7.89	7.95	5.59	7.17	6.36

	Black			Black		
	Male	Female	Total	Male	Female	Total
1967	11.21	14.10	12.66	8.14	11.43	9.72
1968	9.27	14.77	11.93	12.31	11.65	12.01
1969	10.79	13.60	12.20	12.43	14.25	13.34
1970	10.68	10.95	10.81	11.83	13.91	12.87
Total	10.47	13.80	11.90	11.37	13.10	12.21

constant during 1967 and 1968 but rose significantly in 1969 and 1970.

Table 6 helps to elucidate the reasons for these trends. The only population segment with generally increasing prematurity rates over the 4-year period is the black-private group. The generally higher prematurity rates in this group, and the fact that the proportion of CHW births in this group is increasing, account for the increase in overall prematurity rates for the hospital. However, considering the relatively low 1970 mortality rates among the prematures arising from the black-private segment, this increase in prematurity rates apparently has not caused an increase in the overall hospital mortality rate.

Comment

What would happen if all population segments had the 1970 prematurity rate for staff white babies (4%) and the optimal 1970 weight-specific mortality rates shown in table 4? Applying these optimal rates to the births as they occurred in 1970, we derive an overall CHW mortality rate of 4.8/1000. This would compare very favorably with any in the nation, and it provides a realistic goal for both obstetricians and pediatricians serving CHW. If obstetricians can equal their best prematurity rate in all patient groups, and if pediatricians can equal their best weight-specific mortality rates across all population groups, newborn mortality and morbidity at CHW will be among the best in the country.

Acknowledgement

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Continuing Education in Psychiatry in a Community Hospital

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In the spring of 1967 the Department of Psychiatry at Georgetown University, noting the lack of continuing educational programs in psychiatry for non-psychiatric physicians in the Washington area, initiated a series of seminars titled "Clinical Psychiatry for the Practicing Physician.[1] It consisted of 10 weekly 2-hour didactic and case presentation seminars and was attended by a total of 22 physicians. That Fall and again in 1968 a second and third series of "advanced" seminars was offered, and 14 of the original group elected to continue with this program.

These introductory seminars were designed to improve the ability of physicians to manage patients with emotional problems. It was also anticipated that these seminars will alter the physicians' attitudes and capabilities in dealing with their patients' emotional problems.

At the conclusion of this series of "advanced seminars" it was thought that for the program to survive successfully some changes would have to be made. These changes involved recruitment of new physician-student members and program changes, as well as recruitment of faculty personnel. It was at this point that the concept of holding seminars at community hospitals was considered, and subsequently 2 separate groups were formed. One such group was at the Columbia Hospital for Women, and the following is a report of the activities of this group.

Two members of the Columbia staff who had participated in the previous 3-yearly seminars helped to organize the community hospital group. They were joined by 4 other 3-year members, 3 of whom were general physicians and the other a pediatrician. In addition, 6 new members, all obstetricians-gynecologists, 5 of whom were members of the Columbia staff, were added to the group. It is interesting to note that the available places were quickly filled by word of mouth, and a number of other staff members have evinced an interest in joining the group should an opportunity present itself.

This heterogenous group has been meeting at Columbia Hospital for the past 2 years. The seminars are now 1½ hours in length and are held on Wednesday afternoons starting at 5:00 P.M. There are 24 meetings per year divided into a spring and fall series of 12 seminars each. The early first series of introductory seminars at Georgetown had a structured format of literature presentation, case discussion, and patient interviews, with subjects to be covered scheduled before the course began. As participants expressed their own preferences, more flexibility was allowed and the group members focused on problems of most interest to them. At the present time case presentation by a member of the group with discussion by the group including the 2 psychiatrist leaders has become the principle teaching technique employed. The subjects that have been considered are abortion, both as regards the immediate and long-range effect on the patient, frigidity, impotence, homosexuality, family conflicts, behavioral patterns for the young, and hysterical conversion reactions.

The seminar format is ideal for combining the roles of student and teacher, and the mixing of new with older members has proven to be very beneficial. The more experienced students help the newer ones along, and this insures that discussions are sufficiently stimulating to the interest of all. The result of this blending at different viewpoints and levels of expertise has been beneficial to old and new group members alike. Listening to the new participants reminded the experienced members of the significant changes that had occurred in their own attitudes and skills over the preceding years. Hearing how other practitioners were utilizing psychiatric concepts provided a good orientation to new members and gave them courage to attempt to do likewise.

It is now felt by the group and the teaching psychiatrists in view of their experience with this program, admittedly limited, that the highly structured initial experience does not appear to be important. In fact, consistent with Pearson's[2] studies, unstructured experience is actually superior.

It is of interest to note the changes that have gradually taken place in the attitudes and abilities of this group as well as the scope of their practice as they have progressed in the course. Of the physicians who participated in 2 or more series of seminars nearly all experienced a feeling of greater confidence or comfort in dealing with patients. This was most often expressed by such statements as being better able to manage troublesome patients and being a better listener and less judgemental and more supportive with their patients. Several have acquired greater insight into their own feelings and behavior and have noted improvement in their relationships with their family members and/or office staff as well. Almost all members of the group reported that their increased knowledge or skill had favorably affected their practices.

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What motivates physicians to enroll and stay with courses in psychiatry? A few attend in an attempt to deal with their own emotional problems. The majority, however, wish to improve their own interpersonal relationships as a psychotherapeutic goal. Most of these thought that many of their patients had emotional problems, either exclusively or as part of their physical problem yet not suffciently severe to refer them to a psychiatrist. They felt they were personally not as well equipped as they should be and were conscious of disappointments, failures, or mistakes in their practices and felt the need for more understanding of emotional factors if they were to successfully treat, manage, and properly refer their patients. This has become particularly germaine to the obstetrician-gynecologist members of the group as they have found their role as being the primary physician in women's care expanding over the past few years. With the changes taking place in modern medical care the obstetrician-gynecologist finds himself confronted with an ever-increasing demand for his services in this regard, and a better understanding of the emotional components of his patients are of paramount importance in helping him to deal effectively with their problems.

Since one of the most important factors in helping these patients is to give them sufficient time to talk about their problems it is significant that nearly all in the group now set aside time each week for counseling. This is being done not at the expense of time normally spent in seeing patients but by scheduling patients to come at hours when normal office hours are not being held.

The time necessary in this group to initiate significant change in their practice was fairly constant. Some made changes in their practices during the latter part of the second year, but significant modifications were not usually made until the third or fourth year. Many studies have shown that in all types of practice there is often a large segment of the patients in need of emotional support and help. They are not sick enough to need a psychiatrist's care, but they do need the type of aid that any interested practitioner can give them if he or she is willing to spend some time and effort in learning a new technique. Certainly this group at Columbia Hospital feels that they have a much more satisfactory practice both for themselves and their patients than they did before taking the course.

It is the hope of the Department of Psychiatry at Georgetown University that more practitioners will avail themselves of this learning experience and that other groups can be started in community hospitals.

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Do Estrogens Cause Breast Cancer? A Critique

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It is well known to the medical profession that large and increasing amounts of estrogenic substances in various forms and combinations have been prescribed and consumed over the past 3 decades. It is estimated that in excess of 500 pounds of estrogens are being consumed each year.[1] Because of the increasing numbers of women who are given estrogenic preparations for various indications, such as contraception, osteoporosis, and the management of menopausal symptoms, the question of potential harmful side-effects of such preparations becomes of major public health significance.

We are concerned in this review with one of the possibilities referred to as a complication of estrogen therapy, namely, a potential effect of estrogens as a causative factor in the development of breast cancer. The concern is not new. Dr. Hugh Auchincloss[2] in 1936 reported at a staff meeting in New York a case of breast cancer in a patient receiving a new synthetic estrogen preparation known as diethylstilbestrol, with a grave warning that this estrogen might be an etiologic factor.

It should be stated at the outset that no direct conclusive experiment has yet been accomplished. A statistically acceptable experiment would require a prospective study of 15,000 to 20,000 women aged 20 to 39 years on estrogen therapy followed for 4 years in order to obtain a 90% chance of detecting at the 95% probability level an undelayed 2-fold increase in risk.[3] The element of "undelayed" increase in risk should be emphasized, because there is ample precedent for the concept that we might be dealing with a prolonged latent period, as has been demonstrated in the long latency of certain known carcinogens in humans, pointed out repeatedly by Hertz.[3-5] Lacking such a conclusive experiment, the evidence at hand must be carefully scrutinized and weighed in order that we may draw as rational a conclusion as possible.

It has been demonstrated that a number of neoplasms, including breast cancer, can be produced by the administration of synthetic estrogens in laboratory animals.[6-8] An important clinical result of such experiments has been the discontinuance recently of the manufacture of certain birth-control preparations containing chlormadinone, subsequent to the finding of breast neoplasms in beagle dogs given this preparation (this species being known to be particularly susceptible to breast tumors). In relatively limited studies with monkeys given estrogens, no mammary cancers have been reported, although recent reports to the Food and Drug Administration indicate metaplastic changes in the breast tissue of monkeys given ethynerone.

McGuire and Julian [9] have demonstrated 2 types of cytoplasmic estradiol-binding sites in DMBA-induced rat mammary carcinomas. Only the hormone-dependent tumors exhibited the highly specific "A sites", while the much weaker binding "B sites" were present in both autonomous and hormone-dependent tumors.

In normal mammary tissue in vitro, estradiol has been shown to affect the number of cells entering the DNA synthetic phase of the cell cycle, with either stimulation or inhibition, depending upon the concentration of estradiol.[10] McGuire et al[11] noted a 90% reduction in specific estradiol-binding sites in the mammary carcinoma which responds to ovariectomy as compared to the estradiol-sensitive carcinoma which does not respond. They suggest that this reduction in specific estradiol-binding sites does not permit the direct *in vitro* influence of estradiol on DNA replication that exists in normal mammary cells, and this may in part explain the lack of the estrogen control mechanism in their preparation resistant to ovariectomy.

While no conclusive, prospective study has been carried out, a number of retrospective studies of the incidence of breast (and uterine) cancer in patients receiving various forms of estrogen therapy have been reported.

In reports by Mustacchi et al,[12] Wallach et al,[13] and Wilson et al[14] a total of 716 patients were observed for periods of 5 to 8 years while taking estrogens of various types and dosage, with no breast cancer observed. In these studies, only 84 or the 716 women were between the ages of 20 and 40.

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The FDA Report on The Oral Contraceptives, August 1, 1966, summarized reports of material totaling 4,998 patients, studied for a total of 103,692 cycles.[15] There were no mammary cancers and only 3 genital malignancies, during treatment.

In a very recent study Vessey et al[16] interviewed 166 women undergoing biopsy for a benign breast lump and 54 undergoing primary treatment for breast cancer, along with 216 controls selected from the same hospitals and carefully matched for age, marital status and parity. They concluded that there is no evidence that oral contraceptives are associated with an increased risk of breast neoplasia. Moreover, they interpreted their preliminary data as suggesting that these preparations tend to protect against the development of benign lesions.

Byrd and Burch[17] have recently reported on a 100% follow-up for 9 or more years on 511 women given supplemental estrogens after hysterectomy with oöphorectomy, the observed experience totaling over 5,000 woman-years. Breast cancer occurred in 9 patients with no bilateral occurrences and no deaths from breast cancer. These investigators concluded that, for unexplained reasons, there was an apparent delay in the onset of breast cancer among the women on estrogen compared with women in general. They reported that cancer incidence in any site did not exceed one-third of the incidence anticipated in the general population of white women in the United States. In the 96% of the study group accumulated from patients on conjugated estrogens, there was less mortality, lower incidence of all cancers, fewer deaths from breast cancer, and a somewhat diminished incidence of breast cancer, all being statistically significant.

Fechner[18] described the patterns of the carcinoma and the adjacent breast tissue from 5 women who were taking oral contraceptives at the time a breast carcinoma was discovered. These were compared to controls, who were young women with breast cancer not taking oral contraceptives. No gross or microscopic features were recognized as different from the control group, and there was no unusual morphological response. This investigator estimated that approximately 8,500,000 women in the United States took oral contraceptives in 1969. Nevertheless, the frequency of carcinoma of the breast in women under 35 years of age remained at 4% of all breast cancers in his hospital population, which percentage was similar to the incidence in young women before the oral contraceptive hormones became available.

Goldenberg et al[19] described a peculiar morphologic structure seen in breast fibroadenomas in 4 young women taking contraceptive hormone preparations, and later saw 3 more cases. They noted severe epithelial proliferation but no carcinoma-like changes. Follow-up for 10 to 44 months or longer, with 2 patients on continued hormonal treatment, revealed no recurrence.

One of us (D.H.K.) has reviewed the incidence of breast cancer in 4,900 private patients seen in his gynecology practice over a period of 8 years from January 1, 1962 to December 31, 1969. There were 4,000 patients who received no hormones and 900 patients who received various estrogens in the usual therapeutic dose range, with the most usual form and dose being 1.25 mg of Premarin daily.

Of those who had not received estrogen, 11 women developed cancer of the breast as proven by tissue examination. Their ages ranged from 30 to 74 years, with an average age of 46.5 years. Of the group who had received estrogenic hormones for varying periods, 6 patients developed breast cancer. Their ages ranged from 41 years to 56 years, with an average of 49 years. Five had received Premarin in varying dosages, and one had been given Enovid-E. The period of estrogen treatment averaged 3-1/2 years, but 1 patient had received medication for only 9 months. The vaginal smears on all of these patients were interpreted as "Class I" with low estrogen and moderate progesterone effect. One of the patients, who had no estrogenic therapy for 1 year preceding the diagnosis of breast cancer, had a post menopausal smear with no estrogen effect.

It is important to take cognizance of the logical arguments presented by those who caution against the carcinogenic potential of administered sex steroids, as especially represented by Hertz.[3-5] The observation that no breast cancer has been observed in prepubertal females suggests that the breast tissue must be conditioned by endogenous sex steroids before malignancy can develop. The transient stimulation of metabolic activity of metastatic breast cancer by estrogen administration, and the induction of remission in young women with breast cancer by oöphorectomy both point to a stimulatory effect of estrogen on existing breast cancer. On the other hand, a substantial proportion of older women with breast cancer, and, to a lesser extent younger women, undergo regression of breast cancer from the administration of estrogens.

The prolonged latent period noted with many known human carcinogens is worthy of emphasis, and Hueper[20] noted ranges of latency from as short as 2 years in a case of bladder cancer from aromatic amines, to 25 years with thorium dioxide-induced liver cancer and coal-tar-induced skin cancer, and as long as 30 years with Xray-induced skin cancer and radioactive paint-related bone cancer.

An even further extension of the concept of latency has been recently proposed,[21,22] with the indication that stilbestrol administered during pregnancy may result in the subsequent development of vaginal cancer during teenage years in the offspring of such pregnancies. If this concept of *in utero* predisposition to later development of malignancy is verified, it must certainly give one reason for careful consideration of the possibility of such factors in later development of breast cancer.

It has also been pointed out that prolonged periods of pathogenesis, involving many years, have been demonstrated with carcinoma of the cervix and endometrium with cytologic smear techniques. In contrast, we have no clear knowledge of the "pre-clinical" or pathogenic phase and its duration in the case of breast carcinoma and no awareness of the effects of estrogens on this phase.

The reports of the incidence of breast cancer in males receiving high doses of estrogen point strongly to a carcinogenic role for this agent in this setting.[23]

With regard to animal data, as in the case of many other physiologic and pathologic phenomena, species specificity may preclude projection of the findings into clinical consideration. It is reassuring that estrogens have never produced a breast cancer in experimental primates.[24]

While casual clinical observation without meticulous statistical treatment is potentially conducive to erroneous conclusions, millions of women have been receiving estrogens for menopausal therapy for periods ranging from a few months to many years, without any reported increase in the incidence of breast cancer. After 12 years of increasing use of birth control pills there is no convincing evidence that breast cancer is induced, or that the overall incidence of breast cancer in young women is increasing.

In the few studies at hand, as noted above, with observation during long-term administration of estrogens, there has been no demonstrable increase in breast cancer.

The limited number of pathologic examinations, both in breast cancers and in fibroadenomas, have revealed essentially no differences from those noted in patients not receiving estrogenic medications.

Concerned investigators have pointed to the potential effect of estrogens in accelerating the growth of early or quiescent breast neoplasms,[25] but, as pointed out by Rogers[26,27] the issue is not that certain tumors are estrogen-dependent and can be stimulated by estrogen, but whether exogenous estrogens can induce cancer in the breast where none existed previously.

Further time will be required, along with ongoing prospective evaluation, before it can be stated that the widespread use of estrogen-progestogen preparations in younger women does not carry with it an increased risk of breast cancer. Such considerations as the estriol-excretion quotient, found by Lemon[28] to be subnormal in approximately two-thirds of untreated caucasian breast cancer patients, or, as has been suggested, following a large number of women studying their sex steroid profiles to determine an influence on incidence of breast cancer, may illuminate the problem of a role of sex steroids in pathogenesis. The extensive studies of Bulbrook et al,[29,30] indicate a subnormal excretion of androgen metabolites in women who subsequently develop breast cancer.

Assuredly, final clarification of whether or not administered estrogens are a causative factor in the development of breast cancer in the human female has not been attained, and does not appear to be soon forthcoming. Until definitive studies have been achieved, the clinician, as in so many other areas of medical practice, must be guided by reason. The evidence at hand does not suggest that the administration of estrogens, in the presently accepted preparations and dosage, plays a role in the etiology of human breast cancer.

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Laparoscopy

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Laparoscopy began in middle Europe over 70 years ago. It has only been recently, however, that this procedure has gained popularity in the field of gynecology in the United States. This is reflected in our experience at Columbia Hospital for Women where laparoscopy was first introduced 2 years ago and has increased in use to presently represent between 15-20% of our surgical schedule. This report will deal with our experience to date and the techniques utilized.

Numerous terms have been applied to this surgical procedure as it has evolved, including abdominoscopy, ventroscopy, coelioscopy, peritoneoscopy, and pelviscopy. Jacobeus[1] originated the term laparoscopy and it remains the most widely used. Significant events in the present technique are the following:

1878 Nitze developed the cystoscope
1902 Kelling[2] of Germany used the cystoscope to view the peritoneal cavity (in dogs)
1910 Jacobeus[1] of Sweden described laparoscopy and was the first to perform the operation in living humans
1911 Bernheim of the United States, using a proctoscope, viewed the upper abdomen including intragastric ulcerations
1937 Anderson[3] of the United States performed coagulation laparoscopy sterilization
1946 Palmer[4] of France designed an intrauterine probe and stressed the importance of monitoring intraabdominal pressure
1967 Steptoe[5] of England authored the first textbook in English and established a large series of laparoscopies including sterilizations

Patient Selection

Table 1 lists the indications for the use of laparoscopy. Marked obesity, previous surgery, and advanced malignancy may make the procedure more difficult but are not absolute contraindications. Those conditions in which alternate surgical approaches should be made are listed in table 2.

Technique

Patients are usually admitted a few hours prior to surgery having eaten nothing by mouth after midnight. Selected cases may be done on an in-and-out basis or admitted the preceding day. An enema may be given if the patient has significant gas or fecal retention but is usually not necessary. The patient voids just prior to surgery and is catheterized on the operative table if bladder residual remains. Shaving the pubic area is done only if a laparotomy may be performed. The majority of cases are done under general intubation anesthesia although local with meperidine and valium may also be used. The patient is positioned in a modified dorsal lithotomy position with the legs at a 45° angle. The supine position may also be used. The vagina, perineum, and abdomen are 'prepped' and draped to expose an operative field from the umbilicus to the peroneal body. An intrauterine probe is inserted except in cases in which an intrauterine pregnancy may be present.

The abdominal wall is elevated by grasping the paniculus by hand or by applying towel clips at the umbilical margins. A Verres insufflation needle is inserted through the lower border of the umbilicus and directed toward the mid pelvis. Aspiration through the needle at this point is important to rule out perforation of bowel or major blood vessel prior to insufflation. With the needle tip in the peritoneal space, CO_2 is insufflated at 1 liter per minute. Percussion is used to confirm a developing symmetrical tympany, and the intraabdominal pressure is monitored throughout the insufflation. Pressures usually are below 15 mm hg. A total volume of 2 1/2 to 3 liters of gas is usually sufficient to establish a satisfactory pneumoperitoneum. A skin incision corresponding to the trochar size is then made at the needle site and the telescopic trochar inserted. A "Z" tract along the lower midline fascia directed toward the mid axis of the pelvis is used to effect an air seal following the surgery. Appropriate placement is confirmed by noting a rush of air when the trochar valve is opened. The patient is then placed in a 20° Trendelenberg position and the uterine probe manipulated to view the pelvic structures. Trochars for probes or ancillary surgical instruments may

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TABLE 1: Indications for Laparoscopy

1. Investigation of infertility secondary to adnexal disease.
2. Unexplained abdominal or pelvic pain.
3. Primary or secondary amenorrhea associated with developmental anomalies or ovarian dystrophies.
4. Suspected external pelvic endometriosis of limited extent.
5. Suspected ectopic pregnancies.
6. Evaluation of adnexal or pelvic masses.
7. Assessment of chronic pelvic inflammatory disease, particularly its response to prolonged conservative treatment.
8. Some endocrinological syndromes.
9. Differential diagnosis of appendicitis vs. salpingitis.
10. Evaluation of pelvic malignancies and their response to therapy.
11. Cytology of pelvis and peritoneum.
12. Sterilization interval and post-therapeutic abortion.
13. Ovum recovery.

TABLE 2: Contraindication for Laparoscopy

1. Advanced cardiovascular or respiratory disease.
2. Hernias of the abdominal cavity.
3. Generalized peritonitits or a history of it.
4. Intestinal obstruction.
5. Pelvic mass palpable abdominally.
6. Hemorrhagic shock.
7. Chronic tuberculous peritonitis.
8. Advanced malignancy involving the anterior abdominal wall.

be placed in the lower abdomen. Transillumination with the intra-abdominal laparoscope affords identification of large vessels in the parietal abdominal wall. When coagulation procedures are being performed, certain precautions are taken. The diathermy instruments are testes before use. The instrument insulation is checked. Only the laparoscopist himself operates the foot pedals. Coagulation proceeds only while the area being coagulated is under direct vision by the laparoscopist. A clear space between bowel and other vital structures is maintained.

At the completion of the surgery the gas is expressed and the trochar sites closed by a single subcuticular suture. Band-aids are used for dressings.

Postoperatively the patients are given an aspirin or codeine type of analgesic and are allowed to ambulate when reactive. Some patients are discharged after 6-8 hours but most remain until the following morning. The recovery at home is usually accomplished within a week. Lifting is avoided during this period, and the patient is allowed to return to her usual activities in 3 to 4 days.

Columbia Hospital Series

In the past 2 years 931 laparoscopies have been performed at Columbia Hospital for Women. Diagnostic indications accounted for 477 cases and sterilization for 454. Table 3 lists the admitting diagnoses. Ancillary surgical procedures were performed in 559 patients, listed in table 4. Laparoscopy was combined with other surgical procedures including D&C, biopsy of the cervix, endometrial biopsy, polypectomy, cryoconization of the cervix, vein ligation, excision of perineal papillomas, abdominal scar revision, and IUD insertion and removal.

Complications

No surgical deaths occurred in the series. Neither trochar perforation of the bowel, or major blood-vessel, nor thermal injury to the bowel was encountered. Six exploratory laparotomies were performed to evaluate

TABLE 3: Admitting Diagnosis for Diagnostic Laparoscopy

Diagnosis	%
Infertility	32
Pain, abdominal or pelvic	21
Suspected ectopic pregnancy*	9
Adnexal mass†	8
Endometriosis	6
Chronic salpingitis	5
Stein-Leventhol syndrome	4
Amenorrhea	4
Pelvic mass	3
Appendicitis	0.8
Other	6

* 22 of 33 cases required no additional surgery

† 25 of 30 cases required no additional surgery

TABLE 4: Ancillary Surgical Procedures Done by Laparoscopy

Procedure	Number
Sterilization by coagulation and resection*	433
Tubal insufflation	92
Lysis of adhesions	27
Biopsy of ovary	16
Aspiration cytology	15
Aspiration of ovarian cysts	7
Biopsy of peritoneum	5
Coagulation of endometriosis	4
Removal of intra-abdominal IUD	6
Other	11

* Includes 43 done following therapeutic abortion by aspiration.

complications. Three were for adnexal bleeding at the site of tubal sterilization. Serosal injury of bowel requiring only suturing was seen in 1 case. Mesentery perforation with an ancillary trochar and needle puncture of the stomach accounted for the other cases. The needle puncture occurred in a patient with a markedly distended stomach who was not intubated. Wound infections at the trochar sites were rare, even though these areas, particularly the umbilicus, harbor high bacterial counts. Frequently the eyepiece of the telescope comes in contact with the face, but infection does not appear to be a frequent problem. Parietal abdominal-wall emphysema, on the other hand, was frequently encountered until confidence in the needle-puncture technique was accomplished. The CO_2 was rapidly absorbed in a matter of a day or 2 without sequelae.

Discussion

Laparoscopy has proved to us to be a valuable addition to the clinical practice of obstetrics and gynecology. A significant impact of the procedure is evident in the patients admitted because of suspected ectopic pregnancies and pelvic masses. Laparotomy was avoided in 56 of 77 patients in these categories. This resulted in a reduction of recovery time for the patient and a significant help to hospital utilization. A similar reduction in hospital stay was effected in the area of interval sterilization. With proper technique serious complications such as bowel or blood-vessel injury should rarely occur. The surgeon must be thoroughly familiar with the instruments and learn to establish eye and hand coördination before attempting ancillary surgery through the laparoscope.

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Laparoscopy, A Modern Gynecologic Necessity

GRANT P. BAGLEY, M.D.*, HERBERT L. KOTZ, M.D., FACOG†,
AND OSCAR I. DODEK, JR., M.D., FACOG‡

Recent advances in equipment and techniques of laparoscopy have made utilization of this procedure a necessity for the modern practice of gynecology. In this paper we intend to demonstrate how laparoscopy has changed the traditional approach to several common gynecologic problems. Numerous reported series over the past 5 years have shown the usefulness and safety of this procedure, and the indications have become well accepted.[1-6] The savings in patient hospitalization, postoperative discomfort, and expense have been proven.[2-5]

Technique

Standard technique is used as described by Cohen.[3] The Wolf Laparoscope[§] is employed with the Eder Automatic CO_2 insufflator. Eder-Cohen Biopsy Tongs are used for tubal sterilization and biopsies. We have found that removal of a section of fallopian tube at the time of sterilization does not consistently provide an adequate specimen for microscopic examination. Also, the likelihood of encountering minor bleeding is increased when a section of tube is removed. For these reasons we have limited our sterilization technique to cautery and division of the tube.

All procedures are carried out under general anesthesia with endotracheal intubation. Adequate relaxation of the patient is a necessity.

Results

Since the introduction of laparoscopy in our hospital 6 months ago the procedure has been performed 160 times. Slightly less than half of these were for sterilization. Our indications have been the same as those reported in previous series.[2-6]

We think that the incorporation of this operative technique into our armamentarium in a relatively short period of time warrants presentation. The utilization of the laparoscope over the first 6 months of its use is shown in figure 1. The rapid adoption of this technique is evidence of its usefulness and value. Even more striking is the number of gynecologists employing the procedure. Figure 2 shows dramatically the acceptance of the procedure by the attending gynecologic staff.

Prior to including laparoscopy in our armamentarium, culdoscopy was used for diagnosis in most cases of obscure pelvic pain, infertility, and endocrine disorders in which inspection of the pelvic organs was thought justifiable. We now find culdoscopy to be a rarely performed procedure. The use of the laparoscope provides better visualization of pelvic structures and operative procedures are more easily accomplished.

The following cases serve to illustrate how we have utilized this procedure in the solution of unusual

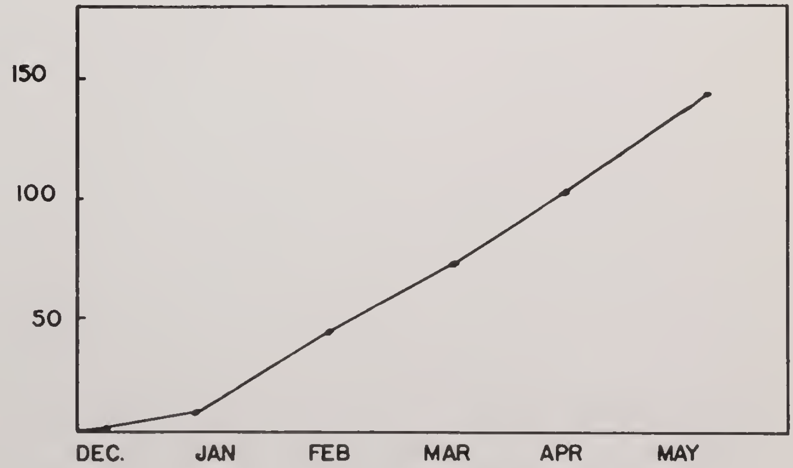

FIG. 1. Number of procedures.

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[§] Lippes Loop. Raritan, New Jersey: Ortho Pharmaceutical Corporation.

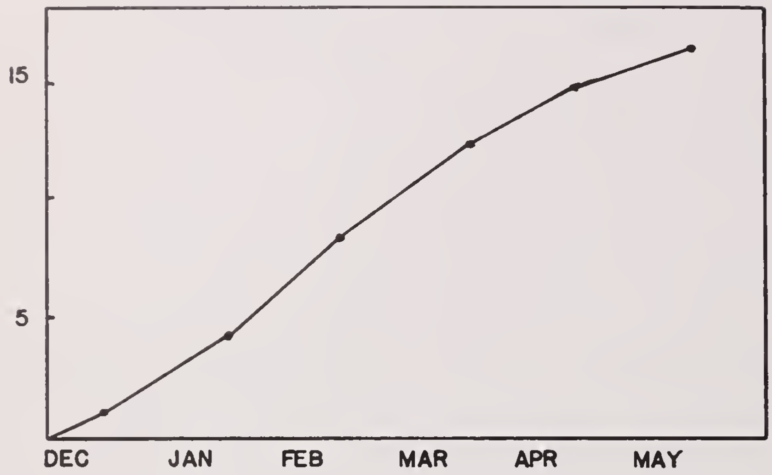

FIG. 2. Number of gynecologists using laparoscopy.

gynecologic problems. They also illustrate the changing manner in which problems are approached when the laparoscope is available.

Illustrative Cases

Case 1. A 27-year-old, gravida 3 para 3 was referred for laparoscopy and removal of an intraabdominal intrauterine contraceptive device. She had an I.U.D. insertion at the time of her 6-week postpartum examination 3 months prior to referral. Speculum examination had subsequently shown an abscence of the I.U.D. string. A flat plate of the abdomen indicated that the device was intraabdominal and lying in the region of the cecum.

Laparoscopy was performed under general anesthesia, and the string from the lower portion of the I.U.D. was seen along the right pelvic brim. This was traced back to filmy adhesions between the cecum and the right pelvic wall. These adhesions were easily divided, and the lower portion of a Lippes Loop*** was seen. This was grasped and the I.U.D. removed with broncoscopy foreign-body forceps which fit through the laparoscope trocar. The patient was discharged from the hospital the following day and recovery was uneventful.

The location and removal of a displaced I.U.D. by laparoscopy has been described by Smith.[7] The dilemma posed by a patient with an I.U.D. displacement provides an excellent use for the laparoscope. Even though I.U.D.'s with non-closed loops are not generally thought to present a great hazard of herniation of bowel and subsequent obstruction, the majority of opinion seems to favor removal if possible.[7] Most patients with a displaced I.U.D. are relatively symptom-free and may be reluctant to undergo major surgery. The alternative approach of laparoscopic examination and removal offers a more attractive solution to the problem. Since tubal sterilization is easily accomplished at the same time as removal of the I.U.D., it should be made available to the patient. We have dealt with 3 additional cases of displaced I.U.D.s. Two of these were removed; 1 from the broad ligament and 1 from adhesions between loops of small bowel. In the 3d case attempted removal of a Lippes Loop from below resulted in the loss of the distal 1 cm of the loop. This was not present in the uterine cavity and was presumed to be in or through the uterine wall. Laparoscopy showed the fragment to be imbedded in the posterior wall of the uterus with the tip under the serosa. Since it was asymptomatic, tubal sterilization was performed and the fragment left undisturbed.

Case 2. A 29-year-old para 3003 came in on 3/25/71 complaining of increasing pelvic and abdominal pain and faintness of approximately 4 hours' duration. The pain began during coitus and became progressively more severe. The pulse rate was 120 and the blood pressure was 110/70. The abdomen was tender in the right lower quadrant. Pelvic examination revealed right adnexal tenderness and a bulging of the cul-de-sac. Culdocentesis was productive of non-clotting blood.

At laparoscopy a ruptured corpus luteum cyst was found. There was no active bleeding, but over 1,000 cc of blood and clots was evacuated by means of suction under laparoscopic visualization. Saline irrigation assured the absence of residual clots.

Corpus luteal hemorrhage is a variation of the usual ovarian physiology. The majority of ruptured corpora lutea result in self-limited bleeding, escaping clinical recognition.[8–9] Even moderately severe hemorrhage may stop without surgical intervention.[9] However, massive hemoperitoneum resulting in shock and even death, though unusual, can occur.[10] Several reports have demonstrated that anticoagulant therapy may increase this danger.[11]

It is our belief that laparoscopic examination should be performed in the presence of demonstrable intraperitoneal hemorrhage of questionable origin to rule out ectopic pregnancy, stop persisting hemorrhage by means of laparoscopic cautery, and evacuate free blood and blood clots which may be significant factors in causing posthemorrhage morbidity and infertility. In this case major surgery was avoided, and a pelvis free of blood clots was obtained by operative laparoscopy.

Case 3. A 24-year-old nullipara came in for confirmation of a suspected pregnancy 3 weeks after a missed

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menstrual period. The history was unremarkable, and no complaints were elicited other than early symptoms of pregnancy. Examination revealed a slightly enlarged uterus with a softened lower segment. There was a large, firm, mobile mass palpable in the region of the right adnexa. She was examined 2 weeks later, and the same firm mobile mass was felt. Laparoscopy was performed to delineate the nature of the mass.

Examination of the pelvic organs with the laparoscope showed a 7 cm freely mobile pedunulated fibroid arising from the uterus at the insertion of the right round ligament. The pedicle was 2 cm in length and was broad-based. No further procedures were carried out.

The diagnostic problem of a suspicious mass in the adnexa discovered during early pregnancy can be resolved with minimum risk by employing laparoscopic visualization.

We have experienced relatively few complications from this procedure. Our single major complication was a postoperative intestinal obstruction. Two patients undergoing tubal sterilization required laparotomy due to difficulties in controlling bleeding, but these occurred early in the series.[1 2] With added experience, bleeding has not been a problem. Two episodes of minor infection from the puncture site responded to local treatment. We have performed the procedure on patients weighing up to 285 lbs. While obesity can make the introduction of the trochars more difficult, the procedure is possible in all but the largest of patients. We have had no case in which we were unable to perform the examination.

With improved equipment and techniques, we feel that laparoscopy is a necessity for the modern practice of gynecology. Since our introduction of laparoscopy into a large private hospital its acceptance has been rapid. The assimilation of the technique into the armamentarium of the attending and resident staff over a short period of time attests to its value. The cases presented demonstrate how the use of laparoscopy provides new approaches to problems in gynecology and do so with the advantages of safety and reduced expense.

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CONTINUING MEDICAL EDUCATION

Digitalis Toxicity

ANTONIO C. DELEON, JR., M.D.*

Digitalis glycoside continues to be an important cornerstone in the management of cardiac failure. By virtue of the drug's pharmacologic effects—especially on the electrical activity of the cardiac fibers, toxicity is a constant, inherent risk in using the drug. A reported overall toxicity incidence of up to 22 percent serves to highlight its importance.

The single most important move in coping with this problem is scrupulous vigilance. All patients placed on digitalis therapy are potential candidates for toxicity! This is especially so in conditions of known increased sensitivity to digitalis such as: 1) the elderly, 2) acute myocardial infarction, 3) myocarditis, 4) patients with chronic lung disease and 5) conditions associated with hypokalemia, hypomagnesemia, hypercalcemia and alkalosis.

Once the possibility of toxicity is considered, recognition is not always an easy task. The gastrointestinal (nausea, vomiting) and visual manifestations are well known but constitute a small percentage of clinical presentation.

The more common, and unfortunately more serious manifestation is the development of cardiac arrythmias and conduction abnormalities. Almost all forms of rhythm disturbances have been described in digitalis intoxication. The problem is compounded by the fact that the intrinsic cardiac disease may precipitate or cause the same rhythm disturbances. The two most common toxic manifestations however, are Ventricular premature contractions and first degree A-V block. The addition to these of Wenchebach phenomenon, A-V dissociation and nodal rhythm account for 78% of arrythmias noted.

In all patients known to be taking digitalis, appearance of the following features strongly suggest digitalis toxicity:

1) Ventricular premature contractions of multifocal origin, fixed coupling, bigemini or trigemini.
2) Appearance of increased PR interval; second and third degree A-V block.
3) Junctional or nodal tachycardia.
4) Regularization of the ventricular rate (nodal rhythm) in a patient in atrial fibrillation.
5) Marked slowing of ventricular rate (50/min. or below) in atrial fibrillation.
6) Atrial tachycardia with block.
7) Bidirectional paroxysmal ventricular tachycardia.
8) Ventricular tachycardia.

There is no single safe and reliable test for digitalis toxicity. One of the simplest (and also the first step in management) is the discontinuance of the drug. The need to wait several days for digitalis levels to significantly decline is however, a drawback especially with the more serious arrythmias. Other tests employed are the Acetyl Strophantidin Tolerance Test, Carotid Sinus Stimulation and by Calcium Chelation using Sodium EDTA Infusion. The currently available methods of blood digoxin level show some promise. Blood levels of 1.1 to 1.4 ng/ml appear to correlate with non-toxic therapeutic levels and valves of 2.1 to 8.7 ng/ml. (Mean 3.3) correlate with toxicity. However, patients especially sensitive to digitalis may be toxic while blood levels are in the "therapeutic range." Thus, while knowledge of blood level is of clinical help, it does not preclude the need to exercise good clinical judgment.

Therapy

1) "Prevention": In conditions of known sensitivity of digitalis, deliverately "undershoot." Slow, gradual digitalization is always preferable when the clinical situation permits. Be aware that alteration in renal function can alter digitalis needs. (i.e. The major pathway for digoxin excretion is through the kidneys.)

2) Early recognition.

3) Stop the digitalis. This by itself should suffice for mild toxicity. (i.e. Occasional Ventricular premature contractions; first degree A-V block).

4) Correct hypokalemia if present. In many instances, Potassium administered even with normal serum potassium levels is beneficial (exception: associated renal failure and high grade A-V block.) The initial use of 40 mEq potassium over a two-hour period by slow IV drip with constant ECG monitoring is preferable.

5) Drugs such as: Procaine amide, Quinidine, Dilantin and Propranolol.

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6) For heart block Atropine, Isuprel and temporary cardiac pacing.

7) Cardioversion only as a last resort for life threatening arrythmias.

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The Medical Model in Psychiatry: Love It or Leave It*

HUMPHRY OSMOND, M.R.C.P., D.P.M.†

Silas Weir Mitchell[1], addressing the American Medico-Psychological Association in 1894, told them that they ran hospitals "which looked and smelled like second-class lodging houses." This could never be said of the pleasing and well designed psychiatric wards of Sibley Memorial Hospital, whose tenth anniversary we are celebrating today. Yet we all know of wards in mental hospitals now, where patients live and sleep fifty or more to a single inclosure, not as in second-class lodging houses, for such places are doss houses.

Rather than dwell on past failures or persent miseries, I shall reflect on some of our future problems. Not to prophesy doom nor to predict the millenium, but to suggest some modest proposals which, costing little or nothing, might make life easier for our patients, their relatives, our staffs and so for us too. It is said that psychiatry is suffering from an identity crisis.[2] If so, what might be the nature of that crisis?

In 1894, Silas Weir Mitchell reproached us, who had been one of the first specialists, for retiring to the country side, becoming landed proprietors and so losing touch with medicine. He could not accuse us of this today. Indeed it sometimes seems that we are about to abandon our sickest patients for excursions into high politics, government, running the country, or even the world. Yet with these schemes, for a nonmedical psychiatry, few of us have made that first step this would prove our sincerity, which would be to remove ourselves officially from medical practice.

About six years ago, Miriam Siegler and I began to make a series of conceptual models for comparing the various approaches to psychiatric illnesses. So far these models are: medical, moral, social, impaired, psychoanalytic, family interaction, conspiratorial and psychedelic.[3,4,5,6] We have studied these models in twelve dimensions, these are: definition or diagnosis, etiology, interpretation of behavior related to the impairment or illness; treatment, prognosis, suicide, function of the hospital or other institution, termination of hospitalization or institutionalization, personnel, the rights and duties of society. We later added a thirteenth which is the history of the model. The strength of the medical model, whatever criticisms there may be of it, derives from it having stood the test of millenia. It works and even its shortcomings are familiar and well charted. Most psychiatrists use it most of the time and all of us use it some of the time. The psychoanalytic model derives from it. It is, however, less clearly related to the family interaction, moral, social and conspiratorial models and it is wholly different from the impaired model discovered by Gordon,[7] some years ago. Laing's[8] psychedelic model in which the ill person is held to be on the road to enlightenment, resembles the view of those who oppose medical treatment on the ground that one either gets well or goes to a better place.

For thousands of years illness has been seen as something which few of us are likely to escape and whose treatment must be governed by customs and rules. One of the most important of these has been that of granting the sick person a special niche absolving him from usual social duties and imposing new and more appropriate ones. This Tallcott Parsons,[9] called the sick role. The importance of this role in psychiatry has not been widely studied, which may have made it hard for us to predict the consequences of adopting models which do not include it. The sick role, and with it the doctor-patient relationship, only obtains in the medical model, not elsewhere.

Those people now called psychiatric patients have had other roles at other times. They have sometimes been considered witches or bewitched an example of the moral model. Witches were held to be morally reprehensible and treated by burning at the stake. There are at least five varieties of the moral model. They appear to be in conflict with each other and this has caused and will cause much trouble for penologists and reformers.

The social model in which the disadvantaged person is held to be the victim of social circumstances seems innocuous and benevolent. "Treatment" consists of improving those circumstances, making a better society. There have been enough revolutions during the last half century for some of our patients to doubt whether another one will be of special help to their illnesses, whatever other benefits may accrue.

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* From an address given at the 10th Anniversary of the opening of the Psychiatric Department of Sibley Memorial Hospital, Washington, D.C.

† Director, Bureau of Research in Neurology and Psychiatry, Princeton, New Jersey.

In the psychoanalytic model, which was originally within the medical model,[10] fathers were held to be particularly damaging. After the mid 1920s, however, due to Melanie Klein's[11] influence, mothers came to occupy that bad eminence and have been there ever since.

Bateson's family interaction model considers the "index patient" (the actual sick person) to be the least ill member of the family, the illness being located not in individuals but in the malfunction of the whole family[12] group. Treatment consists of improving communication within the whole family. But where does one draw the line? Recently the family has been extended to include thirty or forty neighbors.

In the conspiratorial and psychedelic models advocated by Dr. Szasz[13] and Laing, the patient is seen as the victim of a malignant or sick society. Laing seems to suggest that the victim may attain enlightenment and so be a means of social redemption. In the impaired model, the impaired person has to accept the permanently lowered status of impairment.

That justly famous book, "I NEVER PROMISED YOU A ROSE GARDEN,"[14] describes Deborah, a schizophrenic girl, being admitted to a well known hospital where she meets the ward doctor for the first time. She says to him:

> " 'I told you the truth about these things you asked. Now are you going to help me?' 'That depends on you,' he said acidly, shut his notebook and left."

Is this the medical model? If you went to a strange hospital with a grave undiagnosed illness and were told after asking the doctor, "are you going to help me?" and he said, "that depends on you" how would you feel? Throughout the book Dr. Fried, said by the authoress to be based upon Dr. Freida Fromm-Reichmann, wobbles uneasily between the medical and other models. Deborah says to the doctor:

> " 'You're saying what they all say—phony complaints about a nonexistant illness.' Dr. Fried replies: 'It seems to me you are very sick indeed.' Deborah, 'like the rest of them here. . . . . ' Dr. Fried, 'do you mean to ask me if I think you belong here, if yours is what is called a mental illness—then the answer is yes, I think you are sick in this way, but with your very hard work here, and with a doctor working hard with you, I think you can get better.' "

This could be said about any serious illness. Yet a little later on Dr. Fried adds:

> " 'First I want to tell you again that I will not pull away symptoms or sickness from you against your will.' "

This implies that Deborah, whom Dr. Fried has already said is very sick indeed, might not want to get well. It is almost accusing her of being a malingerer. Yet Dr. Fried did not believe that she was malingering, but this ambiguous remark shows that she was employing at least two models which were in contradiction.

In the medical model Deborah is very sick indeed, and it is assumed that she wants to get well. In the psychoanalytic model she is regressed and it is assumed that her natural inclination is to stay regressed. The book shows these model conflicts producing all kinds of secondary problems. Deborah and her fellow patients believe that they have in some mysterious way chosen their illnesses and they are guilty and dejected for making such a stupid choice. Meanwhile their families reproach themselves for having "caused" the illness by some error of upbringing. Much energy is wasted in gloom, despondency and self reproach which might be used in getting well again.

Studies of polio-myelitis show that people blame themselves unmeritedly for such natural catastrophes unless they are absolved by explicit medical reassurance that it is not their fault. How much more easily can this occur in schizophrenia where the etiology is still disputed? There are many different views of this illness—let us ask ourselves then which model is likely to do least harm and most good, for *nil nisi bonum,* (nothing unless good) is an ancient medical rule.

In the moral model the source of trouble lies within the immoral person. The social model locates it within society. The psychoanalytic model attaches error to a parent, usually the mother today. The family interaction model considers the sick person the most healthy member of an even sicker family constellation. The conspiratorial model points to a malignly disposed external agency which one might think would increase the patient's fear and apprehension. The psychedelic model considers schizophrenia to be a benevolent process corrupted by a malevolent society. The impaired model exonerates everyone from blame, but the impaired person must abandon all hope of recovery and accept permanently lowered status. Only the medical model, that old and complex tissue of rights and duties, allows a temporary change of status with the possibility of returning to one's normal social role when recovered. This model has evolved with us for at least forty millenia (forty thousand years). It has soothed the anguish and eased the dying of many millions of our species. The rights and and duties pertaining to the medical model are so old and so familiar that we take them for granted. Since illness can strike at any time it can be life saving to know that we are ill and to behave appropriately. Until a decade or so ago, nearly everyone learned the sick role in childhood, not only as a central participant, but as a member of the supporting cast. Yet due to the triumphs of public health and preventive medicine, well-to-do children now grow up with little or no experience of either filling the sick role themselves or seeing it in others. What effect can this have upon adapting to illness in adult life for one would simply not know the rules of this unfamiliar game?

In medicine the role of the responsible patient, backed by a responsible family, has expanded steadily in recent years. Diabetics are taught to give themselves treatment while those who have not yet been diagnosed are told what symptoms to look out for and report to their doctors if in doubt. Heart, lung, kidney diseases and epilepsy are being tackled on similar lines. Patients and their relatives are running artificial kidneys at home, which was only possible in specialized hospitals a few years ago. Thanks to the genius of the late Bill W., alcoholism has become a serious illness and ceased to be a vice, due as much to the efforts of Alcoholics Anonymous as to those of the APA and the AMA.

While preparing for eye surgery recently, I sometimes wondered how I would have felt if my surgeon had countered my questions with either, "why do you want to know what is wrong with your sight?" or perhaps, "you say you want to know what is wrong with your sight?" or again like Deborah's doctor, replied when I asked what could be done to help me, "that depends on you."

One of the first steps the doctor takes is to name the illness, thus making it more finite and so less terrifying. By naming the illness we show patients their adversary and join them in battle against it. Both patients and relatives must be involved in that battle. This was how Dr. Matthew Baillie, John Hunter's nephew, set about it over one hundred and fifty years ago. A contemporary describes him thus:[15]

> "His manner of explaining the disease, and the remedies recommended, was peculiar to himself, and singularly happy. It was a short compressed lecture. . . in such simple unadorned language as was intelligible to his patients, and satisfactory to his colleagues. Before his time, it was not usual for a physician to do much more than prescribe remedies for the malady, and to encourage the patient by such arguments of consolation as might present themselves to humane and cultivated minds. But as. . . a more curious anxiety began to be observed on the part of the patient to learn everything connected with his complaint, arising naturally from the improved state of general knowledge, a different conduct became necessary in the sick room. The innovation required by the spirit of modern times never could have been adopted by any one more fitted by nature and inclination to carry it into effect than by Dr. Baillie."

Patients have a right to know their diagnosis, for without it they cannot become responsible patients. An unknown illness is profoundly discouraging, for to the patient it suggests that it is unknowable.[16] There are patients in general hospitals who become suicidal after hearing there is "no evidence of disease." Yet when told bluntly that they have that well known and treatable illness, an endogenous depression, great is their relief. Making a diagnosis is an essential part of that contract, linking the doctor to the patient and his family and as with any other important contract, it should be undertaken, in a decorous and ceremonious way. Etiology (causation) does not have to be known for a satisfactory diagnosis. But some naming of the adversary, even an absurd one like PUO (pyrexia of unknown origin), or nearly absurd ones like influenza (the influence) is better than nothing.

By using the medical model in psychiatry, the rights and duties of patients, their doctors and everyone else involved, becomes clearly defined. The blame can only be given to those who fail to fulfill their duties. Respect and consideration is always due to someone who has performed his role properly, that is, done his duty. Because of the great antiquity of the medical model, those rights and duties and the role and goals which accompany them, can be easily defined and understood. They are so old that they might almost be built into our genes. None of the other models is anything like, as well known, or as universal to all humanity.

The atmosphere of blame and recrimination which has polluted psychiatry for much of the last generation appears to be the result of demonstrable error, misunderstanding and confusion. Before the sick role has been assigned to any illness there is a period, short or long, when patients unable to perform their social duties seem aberrant, alienated and even immoral. This strains family relationships and may sometimes cause permanent embitterment. It is our duty as physicians to inform people of this and so prevent illnesses, serious enough in themselves, from being converted into social and personal catastrophes. Psychiatric diagnosis today often does not perform this particular and essential function because it is not communicated to patients and their relatives in a way which secures their approval, cooperation and understanding. This is not because patients are unwilling to listen to us–quite the contrary. Our studies[17] show that they are anxious and grateful for any information we give them. Neither have we lost interest in diagnosis, for the categories and subcategories of psychiatric illness continue to grow endlessly. It is the appropriate social use of diagnosis and our grasp of its effect on the doctor-patient relationship which has evaded us. In view of our avowed concern with human relationships, this is sad.

Some hold that the medical model is necessarily limited to physical matters; but due to the limitations of all treatment, medicine has never been and probably never will be confined to purely physical approaches. The physician's aesculapian authority[18] includes not only technical knowledge but wisdom, compassion and sometimes the capacity to inspire patients with hope and so insure their survival when everything shows that they will die. The clinical art is a demanding one.

Perhaps some of our recent descontent with the medical model arises not from its shortcomings, but from our own ignorance. Familiarity breeds not only contempt but blindness to the obvious. The medical model itself appears to be extremely complex.[19] We need a better understanding of our craft and science today than we did in the past when our achievements were less. A rough and ready reckoning may suffice for sailing ships but it would be fatal for a jet plane.

Over a hundred and thirty years ago, Dr. Samuel B. Woodward at Worcester State Hospital, was using the medical model very skillfully in psychiatry.[20]

> "His intercourse with the sick was so gentle, cheerful and winning that he soon gained their confidence and love. He nourished their hopes of recovery by holding up the bright side of their cases. They anticipated this with pleasure, as their physician and their friend. He recognized the influence of the mind over the physical functions and by his relation of agreeable stories and successful cases of a similar kind of theirs, he animated their hopes."

His results seem to have been as good using his methods as those that we get today in similar illnesses.

One seldom meets patients nowadays who tell of their psychiatrist animating their hopes in this way. Even the admirable Dr. Fried never mentioned "agreeable stories and successful cases." Indeed, when Deborah hears of a patient who has recovered and left the hospital she does not at first believe this. In many autobiographies of patients,[21,22,23,24,25] psychiatrists are seldom depicted giving hope, that best of medicine. When they do so, patients relate this with relief, gratitude and above all, surprise.

This has been a sketch of the central part which the medical model ought to play in psychiatry. I do not think it has done so in recent years. Indeed, some of our colleagues have suggested abandoning it. Before jettisoning our rich inheritance, we should, I believe, think deeply. When we have done this, perhaps the story of Maurice Chevalier's 80th birthday will come to mind. The great French comedian was asked:

> " 'Monsieur Chevalier, how does it feel to be 80?' He paused, stuck out that familiar under lip and answered, 'Well . . . considering the alternatives, very good indeed.' "

Let us consider those alternatives. If then we decide to abandon medicine, in common honesty to those who will no longer be our patients (for patients only exist within medicine) we must take leave of them explicitly, forcefully and unambiguously as in Michael Drayton's great sonnet:

> "Since there's no help, come let us kiss and part! Nay, I have done, you get no more of me; And I am glad, yea, glad with all my heart, That thus so cleanly, I myself can free;"

However, should we decide not to kiss and part, the sonnet ends:

> "Now if thou woulds't when all have given him over, From death to life, Thou mighst him yet recover."

If we are not going to leave medicine and I suspect we haven't got the slightest intention of so doing, then it would be best for our patients and for us too, to love her as truly, wisely, attentively, faithfully and as intelligently as she deserves.

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Divorce Counselling: When a Physician Refers Only One Spouse for Psychotherapy

NEWTON FROHLICH LI.B.*

"It's hard for a free fish to understand what is happening to a hooked one," Dr. Karl A. Menninger once wrote. And his warning applies particularly to a "free" physician when confronted with a "hooked" patient complaining of emotional problems associated with an unhappy marriage. At first blush, the best course might appear to refer the unhappy patient to a psychiatrist or psychologist. But the physician should realize that he-or she-may be introducing to the marriage a new form of "triangle" perhaps as destructive to the marriage as the more notorious form.

This is not to minimize the very real benefits that may and so often do result from needed psychotherapy—benefits to either the individual spouse or the married couple or both. Through counselling, individuals and couples can gain insight, confidence and an ability to cope that are crucial to either a happier marriage or a successful divorce. Moreover, increasingly, the techniques for counselling involve the therapist seeing both spouses, together, alternately, in groups, or with their children and parents. But, in a society in which about one in four marriages end in divorce, psychotherapy must be considered with the full appreciation of the fact that it may serve to expedite rather than prevent divorce.

The married patient who complains about emotional problems is probably sharing those problems with his or her spouse. Whether or not the sharing is helpful, the involvement is still great. A delicate balance, previously struck, is in the process of being disturbed. Sometimes that disturbance is growth, perhaps growth by only the husband or the wife, perhaps by both of them. Sometimes "growth" is just a euphemism for change—in occupation, in interests, in financial status. The husband becomes tired of being the soberminded head of the family: he announces he wants a little fun out of life. The wife says she no longer finds being a housewife satisfying: she wants to get a job, become more self-sufficient financially, and get out in the world. Thomas A. Harris, M.D. views these changes in terms of alteration of the parent-child roles in the marriage.[1] Carl A. Whitaker, M.D. discusses this process in terms of a change in the bilateral psychotherapy which so often motivated the couple to marry in the first place.[2] Whatever the cause for the tension in the marriage, if that tension is not recognized as requiring the joint effort of both the husband and wife to overcome it, there is a good chance that husband and wife, living in America in 1972, will be divorced.

Consider how a husband may feel when he learns that the confidences he has expressed to his wife the night before are being revealed by her to the therapist the next day. Consider the frustrations of the wife who learns that her husband is no longer bothering to talk to her anymore: he has a therapist to talk to who is far more understanding and does not talk back. Obviously, sometimes, the entry of only one of the spouses into therapy cannot be helped, and in fact, is imperative. But the indiscriminate referral of a husband or a wife to psychotherapy without appreciation of the consequences to the marriage is regrettable. As Whitaker and Miller have written:

> "We suggest that in a circumstance where divorce impends the ordinary and customary styles of reacting to an appeal for help characteristic of general psychiatric practice may be inappropriate, ineffectual, or, at worst, substantially detrimental."[3]

None of this is to say that marriage counselling is being viewed with deprecation. A well-intentioned physician refers a patient for psychological counselling. Often the motivation on the part of the patient, or the doctor, is avowedly to save the marriage. But in the end the patient becomes aware that his or her marriage is beyond help. They learn why their marriage failed and what they can do to avoid repeating the same mistakes. They are able to change their focus from despair and failure to the possibility of recreating a new life for themselves. They discover that their emotional bombs have been disarmed privately in a doctor's office rather than being allowed to explode in the courtroom. They become able to bring to the process of getting the divorce the necessary objectivity and coolness.

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Today, the legal processes of divorcing no longer provide the great deterrent to dissolving a marriage that they once did. The process no longer adds "insult to injury." When a couple concludes that their marriage is dead, they can divorce decently, relatively inexpensively and privately. Not painlessly, but at least with a reasonableness they never dreamed they would find in the divorce laws. Of course, there are always the pathological cases: the couples, or the husband or wife, who is unable to resist contesting the financial or personal aspects of their divorce in the courtroom. Often, these are the husbands who blame the loss of a meaningful relationship with their children on the divorce system rather than on themselves. Or they are the wives who blame their worsened financial condition on a miserly ex-husband or on a lawyer who did not know how to bring him to his knees, rather than on facing, realistically, the circumstances of their life. Sometimes, of course, fighting in court about alimony or custody is not avoidable. It does take two to negotiate or one of the parties has real reason to challenge the fitness of the other to take care of the children. Moreover, litigation can be an effective bargaining lever. But these contested cases—whether justified or not—are the minority, probably less than ten per cent of the more than 500,000 divorces obtained each year in the United States.

What of the other 90 per cent? They are negotiated. What is negotiated divorce? It is a process, largely legitimized by an unwritten law of divorce, that is recognized, legalized and encouraged in every state in the union and the District of Columbia. It is a process by which a husband and wife—with the help of their lawyers—quietly and privately discuss the problems of dissolving their marriage: Should they separate? Should they divorce? Can they reconcile? Can psychological or psychiatric counselling help? Who shall have custody of the children? On what days should be the visitations of the husband with the children? How much child support should he pay? Should he also pay alimony to the wife? Who gets the house and the furniture? Should any of it be sold? What insurance should be continued? Who will pay for the college education of the children?

The divorcing husbands and wives answer these questions through the process of bargaining and compromise.[4] Their lawyers guide them by advancing each side's best arguments, by using such leverage as each can muster and, finally, by reducing their agreements to writing in a document called a Separation Agreement. This Agreement authorizes the husband and wife to separate and live apart from each other and sets out in black and white their comprehensive plan for the future operation of one family under two roofs. It covers such matters as the bank accounts, the children, support and alimony, college education, life insurance, medical bills and summer vacations. It can maximize the tax deductibility of the financial package if the husband is in the high income tax brackets. It can create a scheme of escalation and de-escalation provisions if the husband's income fluctuates widely. It can deftly control potential friction with well-drafted visitation clauses. It can even attempt to pay for college education expenses with largely tax dollars.

Even the grounds for obtaining the actual decree of divorce once the Separation Agreement is signed can either be simple mutual consent or some other variety of "no-fault" divorce. So, when an unhappy spouse consults a physician—or anyone for that matter—the well-intentioned advice-giver should beware that he may be unwittingly sending that spouse toward a fast moving conveyor belt in which there are very few obstacles to prevent the dissolution of the marriage. And psychotherapy—particularly when administered to only one spouse in a marriage—may oil the wheels.

What Whitaker and Miller, *supra*, have said bears repeating:

> "We believe that psychiatric intervention when divorce impends cannot be regarded as a routine matter. The therapist should view his entrance into the situation as calling for his broadest perspective. He must see what is going on between him and his patient, already a difficult task. Even more than that, he must see what is implied by the fact that he and his patient are sitting down together without the mate. Any move by the therapist that discounts the significance of the marriage may be unexpectedly influential. Thus, the ordinary medical system of replying affirmatively to a request for help by one person in a marriage, excluding the other, may in effect be an intervention favoring divorce.
>
> "In working with a married individual, or with a couple who contemplate divorce, the therapist confronts a system under stress. He would do well, ordinarily, to respect the marriage as a continuing fact until the legal divorce is complete. No matter what the degree of complexity, no matter how seemingly collapsed the patient's marriage is, the therapist should not presume

> to discount its ongoing power, its possibility of resurrection, its beating heart. If there is to be a death certificate, the trial judge of a divorce court of record is the one to sign the document."[3]

At that crucial point, when they might have decided to work on the marriage rather than prepare for divorce, whether they turn *together,* as husband and wife, for professional help, or separately, as individual potentially unmarried people, for divorce counselling, may very well be decided by what approach the physician takes, how he goes about referring—and to whom he refers—the unhappy patient for psychotherapy.

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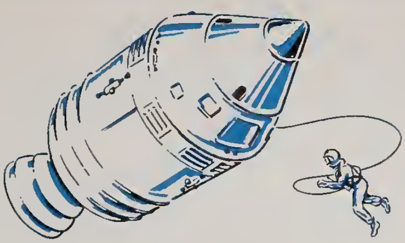

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Warnings: *Drug Dependence:* Physical and psychological dependence and abuse have occurred. Chronic intoxication, from prolonged use and usually greater than recommended doses, leads to ataxia, slurred speech, vertigo. Carefully supervise dose and amounts prescribed, and avoid prolonged use, especially in alcoholics and addiction-prone persons. Sudden withdrawal after prolonged and excessive use may precipitate recurrence of pre-existing symptoms (e.g., anxiety, anorexia, insomnia) or withdrawal reactions (e.g., vomiting, ataxia, tremors, muscle twitching, confusional states, hallucinosis; rarely convulsive seizures, more likely in persons with CNS damage or pre-existent or latent convulsive disorders). Therefore, reduce dosage gradually (1-2 weeks) or substitute a short-acting barbiturate, than gradually withdraw. *Potentially Hazardous Tasks:* Driving a motor vehicle or operating machinery. *Additive Effects:* Possible additive effects between meprobamate, alcohol, and other CNS depressants or psychotropic drugs. *Pregnancy and Lactation:* Safe use not established; weigh potential benefits against potential hazards in pregnancy, nursing mothers, or women of childbearing potential. Animal data at five times the maximum recommended human dose show reduction in litter size due to resorption. Meprobamate appears in umbilical cord blood at or near maternal plasma levels, and in breast milk at levels 2-4 times that of maternal plasma. *Children Under Six:* Drug not recommended.
Precautions: To avoid oversedation, use lowest effective dose, particularly in elderly and/or debilitated patients. Consider possibility of suicide attempts; dispense least amount of drug feasible at any one time. To avoid excess accumulation, use caution in patients with compromised liver or kidney function. Meprobamate may precipitate seizures in epileptics.
Adverse Reactions: *Central Nervous System:* Drowsiness, ataxia, dizziness, slurred speech, headache, vertigo, weakness, paresthesias, impairment of visual accommodation, euphoria, overstimulation, paradoxical excitement, fast EEG activity. *Gastrointestinal:* Nausea, vomiting, diarrhea. *Cardiovascular:* Palpitations, tachycardia, various forms of arrhythmia, transient ECG changes, syncope; also, hypotensive crises (including one fatal case). *Allergic or Idiosyncratic:* Usually after 1-4 doses. Milder reactions: itchy, urticarial, or erythematous maculopapular rash (generalized or confined to groin). Others: leukopenia, acute nonthrombocytopenic purpura, petechiae, ecchymoses, eosinophilia, peripheral edema, adenopathy, fever, fixed drug eruption with cross reaction to carisoprodol, and cross sensitivity between meprobamate/mebutamate and meprobamate/carbromal. More severe, rare hypersensitivity: hyperpyrexia, chills, angioneurotic edema, bronchospasm, oliguria, anuria, anaphylaxis, erythema multiforme, exfoliative dermatitis, stomatitis, proctitis, Stevens-Johnson syndrome; bullous dermatitis (one fatal case after meprobamate plus prednisolone). Stop drug, treat symptomatically (e.g., possible use of epinephrine, antihistamines, and in severe cases corticosteroids). *Hematologic:* Agranulocytosis and aplastic anemia (rarely fatal), but no causal relationship established. Rarely, thrombocytopenic purpura. *Other:* Exacerbation of porphyric symptoms.
Usual Adult Dosage: 1200 to 1600 mg daily, in three or four divided doses; doses above 2400 mg daily not recommended.
Overdosage: Suicidal attempts with meprobamate, alone or with alcohol or other CNS depressants or psychotropic drugs, have produced drowsiness, lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse, and death. Empty stomach, treat symptomatically; cautiously give respiratory assistance, CNS stimulants, pressor agents as needed. Meprobamate is metabolized in the liver and excreted by the kidney. Diuresis and dialysis have been used successfully. Carefully monitor urinary output; avoid overhydration; observe for possible relapse due to incomplete gastric emptying and delayed absorption. REV. 10/71

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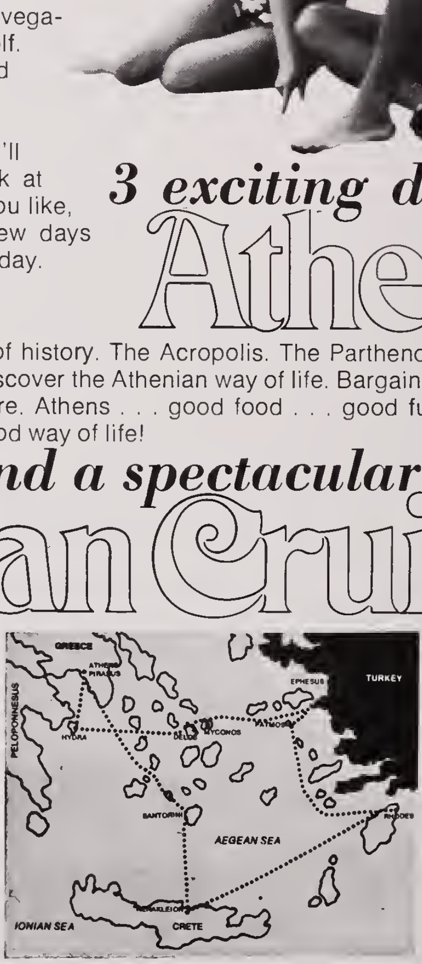

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COMMITTEE REPORT

Water Quality and Supply in the Greater Washington, D. C. Area

The sub-committee on water pollution of the Medical Society's Environmental Health Committee has been studying the problem of water supply and water quality as it pertains to the Metropolitan Washington area. This communication is excerpted from a full report of the sub-committee to the Environmental Health Committee, which was subsequently transmitted to the Executive Board of the Medical Society. It is published in this issue of the Annals to inform the Society membership of the dimension of the various problems particularly as they pertain to the health of the community.

This report is based on data presented at a symposium on the water problem sponsored by the Medical Society in July 1971, on testimony obtained during various hearings conducted by the Interstate Commission on the Potomac River Basin, and on data from the National Capital Region Water and Waste Management Report authored by the Environmental Protection Agency in conjunction with a review of the Potomac River Water Quality Network statistics for 1968 through 1970. Numerous pertinent scientific communications were reviewed as well, and references are appended at the end of this article.

> "Fortunately, the Potomac River turns over rapidly, as opposed to Lake Erie for instance, so that our problem is by no means irreversible."

Credit is also due to Mr. Malcolm Hope of the District of Columbia Department of Human Resources, who serves as technical advisor to the Environmental Health Committee, for his help in compilation of data.

In addition to the problems of water quality and supply, this report considers the status of our drinking water and the sanitary conditions of our swimming pools.

Potomac River Pollution

Briefly, water pollution seems to result from introduction of sewage rich in phosphates, carbon, nitrogen, etc., which stimulate growth of blue-green algae existing symbiotically with bacteria. The latter furnish carbon dioxide to the algae which in turn provide oxygen for the bacteria, resulting in depletion of dissolved oxygen, which may decline to an amount insufficient to maintain marine life. Currently several indices of water pollution are measured. They include the biochemical oxygen demand (BOD, synonymous with organic material), concentrations of various nutrient ions (phosphates, nitrates), presence of toxic substances (arsenicals, mercurials, halogenated pesticides, etc.), content of dissolved nitrogen and concentration of coliform organisms. Whether other parameters of pollution should be measured has not yet been established.

The Potomac River should be cleaned up to protect water quality for recreation purposes and to aid in wildlife preservation. Furthermore, pollution of the Potomac upstream may have deleterious effects on seafood quality in the Bay area. Fortunately, the Potomac River turns over rapidly, as opposed to Lake Erie for instance, so that our problem is by no means irreversible.

The facts concerning Potomac River pollution in our jurisdiction can be enumerated as follows: 1) raw sewage is dumped into the Potomac River at a point known as the Georgetown Gap. This has resulted from failure to connect the so-called Dulles intercept with sewage lines going to the Blue Plains treatment center. Final hookup has been postponed until the Three Sisters bridge construction issue is settled, since its construction would probably result in interruption of the sewage lines. 2) Raw sewage also enters the Anacostia River and the Potomac River at Bolling Field because current Blue Plains sewage treatment capacity of 240 million gallons a day is exceeded by at least 30 million gallons daily due to overflow from the suburban Montgomery and Prince Georges Counties. 3) Following heavy rains, flow from storm drains into the Potomac River and its tributaries is rich in pollutants, particularly coliform bacteria. 4) As a consequence of items one through three, the chief type of pollution between Chain Bridge and Haines Point is a high bacterial count resulting from sewage overflow; from Haines Point to Piscataway Creek pollution results in low dissolved oxygen content. This is due to effluent from waste water treatment facilities. From Piscataway Creek to Maryland Point (approximately 30 miles) the problem is nuisance algal growth resulting from nutrients in waste water discharges. Sewage overflow high in bacteria and low in dissolved oxygen concentration enters the Anacostia River. 5) Prior to 1969 when continuous disinfection of waste water was begun, coliform bacterial densities of 50,000 mean probable number (MPN) per 100 ml were frequently encountered at the Woodrow Wilson Bridge checkpoint. This is considerably above the minimal standard for coliforms of 240 MPN per 100 ml. With institution of treatment, the number of coliforms has been reduced to 1000 MPN per 100 ml. During high run-off periods following storms, over 5000 organisms MPN per 100 ml have been detected in the upper estuary and Anacostia River. 6) The minimum content of dissolved oxygen acceptable is 5.0 mg/L but levels below 2.0 mg/L have been encountered during the summer months over considerable stretches of the river. 7) Salmonella and other intestinal pathogens have been recovered from the Theodore Roosevelt Island checkpoint.

At present, the problem of the Potomac estuary is escalating because of the population increase in the metropolitan D. C. area, estimated to be near 7 million people by 2000 A.D. Treatment facilities in our jurisdiction are obliged to clean up a 25 mile segment of the Potomac River. The updating of the Blue Plains facility for primary treatment and sludge processing is scheduled to be completed by May 1973. Fortunately, construction is currently on schedule. At present 60-70 per cent of the BOD, 15 per cent of N_2, and 15 per cent of PO_4 are removed. A secondary treatment plant, scheduled for completion by 1974, will achieve 90 per cent BOD removal, and 90 per cent phosphate removal. By 1976 completion of a tertiary treatment plant will remove 96 per cent of BOD and phosphates and by 1977 85 per cent of sewage nitrogen will be eliminated. A 309 million gallon per day capability for sewage processing will exist under the present design. However, liquid waste from burgeoning suburbs will require other treatment facilities apart from Blue Plains.

Problems which might be anticipated are as follows: whereas phosphates are essential nutrients in some bodies of water, in others they have not been demonstrated to be rate limiting for algal growth. Rather, carbon which is subsequently converted to bicarbonate may be a greater determinant of algal growth. Hence removal of PO_4 may not result in diminished algal growth. 2) Some sewage treatment plants have been guilty of aerosolizing coliform bacteria downwind from the plant for a distance of one mile. This is a potential health hazard. 3) Current sewage treatment does not preclude problems of heavy metals, toxins and pesticides. These are frequently concentrated within algae as well as suspended in the stream itself. Fish and wildlife who consume the algae or drink the particulate material become contaminated and represent a potential health hazard.

> "Salmonella and other intestinal pathogens have been recovered from the Theodore Roosevelt Island checkpoint."

Water Supply

It has long been speculated that a day might come when the amount of water extracted from the Potomac River for community use would exceed river flow. Such an instance nearly occurred during the drought of 1966. Approximately 75 per cent of the area's consumption is derived from the Potomac River. The average peak winter flow is 7 billion gallons daily, whereas the average daily utilization is 380 million gallons. This latter figure undoubtedly will increase as the population of the metropolitan basin increases. During the summer, flow frequently decreases toward this 380 million gallon a day figure. The average daily per capita water consumption in the United States was approximately 110 gallons 20 years ago. Today it is 142 gallons. Per capita intake in 11 large metropolitan communities is estimated between 132 and 205 gallons daily. The metropolitan D. C. community consumes 136 gallons per capita daily. This is mostly for domestic and municipal usage as little industry exists here. Current patterns of water consumption suggest that increased use is not just related to population increases. Rather each of us tends to consume more. This per capita increase can be attributed to affluence of the community as much as anything. Water is cheap, and wasteful water utilization is common. In one suburban jurisdiction the metered rate of 5.54 per 100 gallons amounts to a 15¢ per ton assessment. A better bargain than that is hard to find. Examples of use include the following: flushing a toilet consumes seven gallons, a shower utilizes 25 gallons, and running a washing machine requires 50-70 gallons depending on the appliance.

Current plans of the Council of Governments call for marked curtailment of water usage on a voluntary basis when river flow rates fall between 550 to 575 million gallons a day, or when daily use is within 100 million gallons of this flow rate. Vigorous enforceable restrictions take place when flow decreases to 525 million gallons daily or when daily consumption is within 50 million gallons of this flow rate.

The Army Corps of Engineers is scheduled to begin construction of a tap on the upper estuary which could remove approximately 100 million gallons a day for recycling and reusage. Since the Potomac is a tidal stream, it can be anticipated that this might be associated with the movement of waste water toward the estuarial pump. The site of this tap is 2.5 miles north of Roosevelt Island, where salmonella species of bacteria have been recovered. Hence any estuarial tap calls for greater community surveillance to prevent contamination and avoid a municipal outbreak.

Besides recycling other solutions to the problem are needed. These include upstream storage through construction of multi-purpose dams and the possibility of tapping into pipe lines from other water suppliers to create greater cross linkage. These could be employed in emergency situations since water stored at the Bloomington Reservoir, for example, is 21 days away. Additional solutions to this supply problem entail further research into water consumption patterns and ways to alter these, particularly during periods of low flow. One might consider changing meter rates at these times. Certainly updating of plumbing facilities in new construction should be encouraged since newer toilet facilities can operate satisfactorily on two gallons per flush rather than the present seven gallons. Still more attractive would be an entirely new concept of waste disposal based on pressure to achieve satisfactory sewage flow rather than large volumes of water to achieve such flow.

Status of Our Drinking Water

Apparently the discoloration of our drinking water which has been a problem in the past is a function of stagnant or reverse flow in the system occurring during periods of peak utilization. This leads to eddy currents which loosen rusty material in the pipes imparting a brown color to the water. There is no evidence of bacteriologic contamination at this or any other time.

The degree of viral contamination in our municipal drinking water is unknown. Preliminary studies conducted in the Northeast Water Quality laboratories in Rhode Island have shown the following viruses added in vitro to Potomac River water can grow and flourish. These include many different enteric viruses such as Coxsackie, Echo, Adeno, Polio, etc. Furthermore, to kill these viruses often requires two hours of exposure to chlorinated water. Adequate disinfection requires suitable combination of contact time and concentration of chlorine. At pH 7.8 a chlorine concentration of 0.5 mg/L takes approximately 40 minutes to kill 99.9 per cent of viruses added in vitro. Other techniques of water treatment such as stagnation and flocculation are also apparently effective against the majority of viruses. Plans for testing the Potomac River at various sites for viral contamination in situ, as well as sampling our drinking water as it comes out of the tap, are under consideration as soon as sampling and viral assay methods are adequately determined.

Contamination of municipal water supplies in Pennsylvania and Iowa by polio virus and hepatitis virus has occurred remotely in the past, but recently most hepatitis epidemics have been attributed to faulty wells or drinking fountains.

Swimming Pools

At present 200 public pools, operating mainly in the summer months, are currently under surveillance. Testing includes detection of coliforms, determination of chlorine residual, as well as pH and clarity of pool water in accordance with the American Public Health Association standards. Although other communities have found on random sampling that the American Public Health Association standards were often inadequate and that pools were frequently contaminated and contained a low chlorine residual, this has not been apparent in random sampling of District of Columbia pools. Strict educational programs for pool operators are in effect, and at present no definite epidemic of infectious (viral or bacterial) origin has been related to swimming pool usage in the last five years.

Summary

The problems of Potomac River pollution and satisfactory water supply as well as considerations of our drinking water and swimming pools have been outlined. The Medical Society feels that such information may be of use to Society members in responding to queries from their patients, and as educational to its members, so that they can intelligently evaluate proposed solutions to these problems.

STUART H. DANOVITCH, M.D.
Chairman
Environmental Health Committee

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d.c. MENTAL HEALTH ADMINISTRATION

The Developmental Services Center

Putting Childhood Back Into the Child

The biggest, shaggiest dog they had ever seen greeted Michael and his mother as they entered the District of Columbia Mental Health Administrations' Developmental Services Center. The dog sided up to Michael's leg and gave him a friendly nuzzling. Michael slammed the tightest fist he could make onto the dog's head and screamed several very loud curse words at the dog.

Michael is not brain damaged, though his I.Q. of 75 is a bit lower than that of most thirteen year olds. His problem is larger than that: He has never had a childhood. At least not the kind people like to think about when they use the word.

His problems began when he entered school. He did not learn to read at the first grade level, but when the time came he was promoted to the second grade. As nearly any psychologist will tell you, had he been held back a grade while his peers advanced he could have been psychologically damaged. His peers began reading at the second grade level and even began learning rudimentary arithmetic. The world of symbols were still new and foreign to Michael. He couldn't integrate them conceptually. In fact, the strange symbols in the first grade reader were still a mystery to him, as were those comprising the letters in his name. Now he was getting less practice with them than ever.

Nonetheless, at the end of his second year in school he was again advanced with his peers to the third grade even though he had not mastered his first grade reader, and certainly not any of the arithmetic concepts. The school system could not afford special tutors for Michael and his parents were interested in other things. Like how to handle Michael's increasingly antisocial behavior at home and in the neighborhood.

By the time Michael was staffed at the Developmental Services Center, he had been tightly bound to his age peers for six years at school and his incompetent scholastic performance constantly compared with theirs. He, like they, had often set goals for himself that involved reading, writing or arithmetic. Unlike those of his peers, his goals were never met. He didn't have the skills. The frustrations mounted daily. The anxiety of being "different" mounted with them while his self concept took an opposite turn and decreased dramatically.

Michael doesn't want love now, not even from a dog. It only reminds him of how unlovable he really is. As unlovable as all total failures are. And he no longer accepts the simplest challenges. He can no longer tolerate the threat of another failure. Another goal that cannot be met.

The staff at the Center are ready for Michael. They have 40 children with problems very similar to his, 72 per cent of whom are male, with ages ranging from 11 to 16 years and averaging 14 years. They have treated over 200 such children since the Center opened in April 1967. Like the others, Michael can expect to stay from 6 to 18 months, depending on the speed of his progress.

He will receive an indepth interview from a social worker, a medical checkup, a more thorough psychodiagnostic workup than most problem children ever receive anywhere, and an educational achievement testing to determine where he is academically. The staff will then come together to determine his program. The same staff group will meet once a month to determine his progress and reevaluate his ongoing program.

The purpose of his program will be to teach him fundamental living skills. He must relearn goal orientation, therefore simple goals will be set for him. Goals that he can reach with his current capabilities. He will receive external rewards—tokens with which he can buy things at the Center's store—until the internal reinforcers take over. Gradually the difficulty of the goals will be increased, the external rewards continuing. The staff will also begin rewarding Michael with tokens each time he sets his own goals. Counselors help him determine which goals are realistic and which he is not yet ready to meet. In this way negative reinforcers are kept at a minimum as unnecessary failures are avoided, and Michael learns the emotionally redeeming art of realistic goal setting again.

Michael will even be taught a skill that most people in contemporary America never learn:

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Pre-Sate ®
(chlorphentermine HCl)

CAUTION: Federal law prohibits dispensing without prescription.

Indications: Pre-Sate (chlorphentermine hydrochloride) is indicated in exogenous obesity, as a short term (*i.e.*, several weeks) adjunct in a regimen of weight reduction based upon caloric restriction.

Contraindications: Glaucoma, hyperthyroidism, pheochromocytoma, hypersensitivity to sympathomimetic amines, and agitated states. Pre-Sate (chlorphentermine hydrochloride) is also contraindicated in patients with a history of drug abuse or symptomatic cardiovascular disease of the following types: advanced arteriosclerosis, severe coronary artery disease, moderate to severe hypertension, or cardiac conduction abnormalities with danger of arrhythmias. The drug is also contraindicated during or within 14 days following administration of monamine oxidase inhibitors, since hypertensive crises may result.

Warnings: When weight loss is unsatisfactory the recommended dosage should not be increased in an attempt to obtain increased anorexigenic effect; discontinue the drug. Tolerance to the anorectic effect may develop. Drowsiness or stimulation may occur and may impair ability to engage in potentially hazardous activities such as operating machinery, driving a motor vehicle, or performing tasks requiring precision work or critical judgment. Therefore, such patients should be cautioned accordingly. Caution must be exercised if Pre-Sate (chlorphentermine hydrochloride) is used concomitantly with other central nervous system stimulants. There have been reports of pulmonary hypertension in patients who received related drugs.

Drug Dependence: Drugs of this type have a potential for abuse. Patients have been known to increase the intake of drugs of this type to many times the dosages recommended. In long-term controlled studies with high dosages of Pre-Sate, abrupt cessation did not result in symptoms of withdrawal.

Usage In Pregnancy: The safety of Pre-Sate (chlorphentermine hydrochloride) in human pregnancy has not yet been clearly established. The use of anorectic agents by women who are or who may become pregnant, and especially those in the first trimester of pregnancy, requires that the potential benefit be weighed against the possible hazard to mother and child. Use of the drug during lactation is not recommended. Mammalian reproductive and teratogenic studies with high multiples of the human dose have been negative.

Usage In Children: Not recommended for use in children under 12 years of age.

Precautions: In patients with diabetes mellitus there may be alteration of insulin requirements due to dietary restrictions and weight loss. Pre-Sate (chlorphentermine hydrochloride) should be used with caution when obesity complicates the management of patients with mild to moderate cardiovascular disease or diabetes mellitus, and only when dietary restriction alone has been unsuccessful in achieving desired weight reduction. In prescribing this drug for obese patients in whom it is undesirable to introduce CNS stimulation or pressor effect, the physician should be alert to the individual who may be overly sensitive to this drug. Psychologic disturbances have been reported in patients who concomitantly receive an anorexic agent and a restrictive dietary regimen.

Adverse Reactions: Central Nervous System: When CNS side effects occur, they are most often manifested as drowsiness or sedation or overstimulation and restlessness. Insomnia, dizziness, headache, euphoria, dysphoria, and tremor may also occur. Psychotic episodes, although rare, have been noted even at recommended doses. **Cardiovascular:** tachycardia, palpitation, elevation of blood pressure. **Gastrointestinal:** nausea and vomiting, diarrhea, unpleasant taste, constipation. **Endocrine:** changes in libido, impotence. **Autonomic:** dryness of mouth, sweating, mydriasis. **Allergic:** urticaria. **Genitourinary:** diuresis and, rarely, difficulty in initiating micturition. **Others:** Paresthesias, sural spasms.

Dosage and Administration: The recommended adult daily dose of Pre-Sate (chlorphentermine hydrochloride) is one tablet (equivalent to 65 mg chlorphentermine base) taken after the first meal of the day. Use in children under 12 not recommended.

Overdosage: Manifestations: Restlessness, confusion, assaultiveness, hallucinations, panic states, and hyperpyrexia may be manifestations of acute intoxication with anorectic agents. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension, or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Fatal poisoning usually terminates in convulsions and coma.

Management: Management of acute intoxication with sympathomimetic amines is largely symptomatic and supportive and often includes sedation with a barbiturate. If hypertension is marked, the use of a nitrate or rapidly acting alpha-receptor blocking agent should be considered. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendations in this regard.

How Supplied: Each Pre-Sate (chlorphentermine hydrochloride) tablet contains the equivalent of 65 mg chlorphentermine base; bottles of 100 and 1000 tablets.

Full information available on request.

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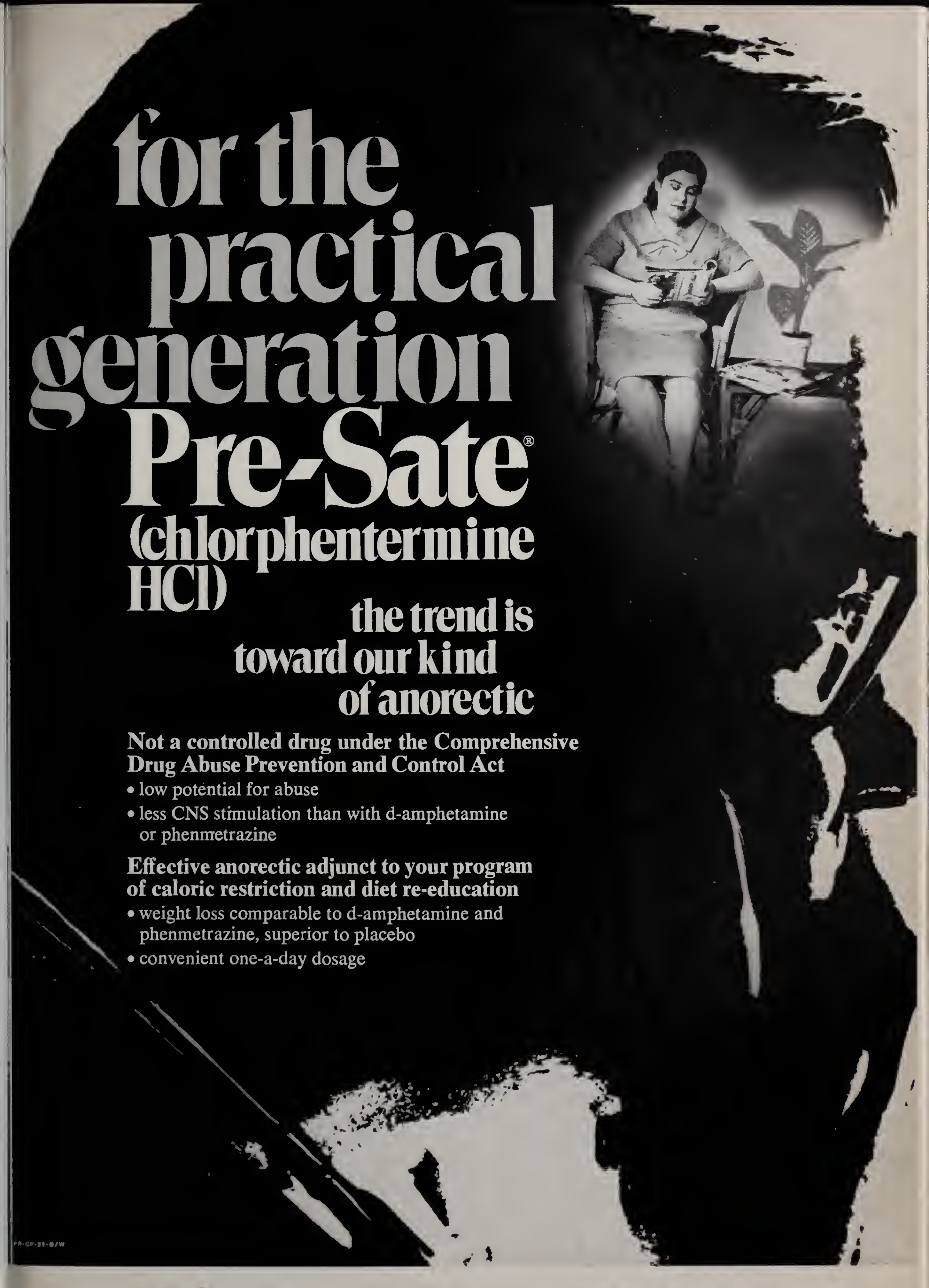

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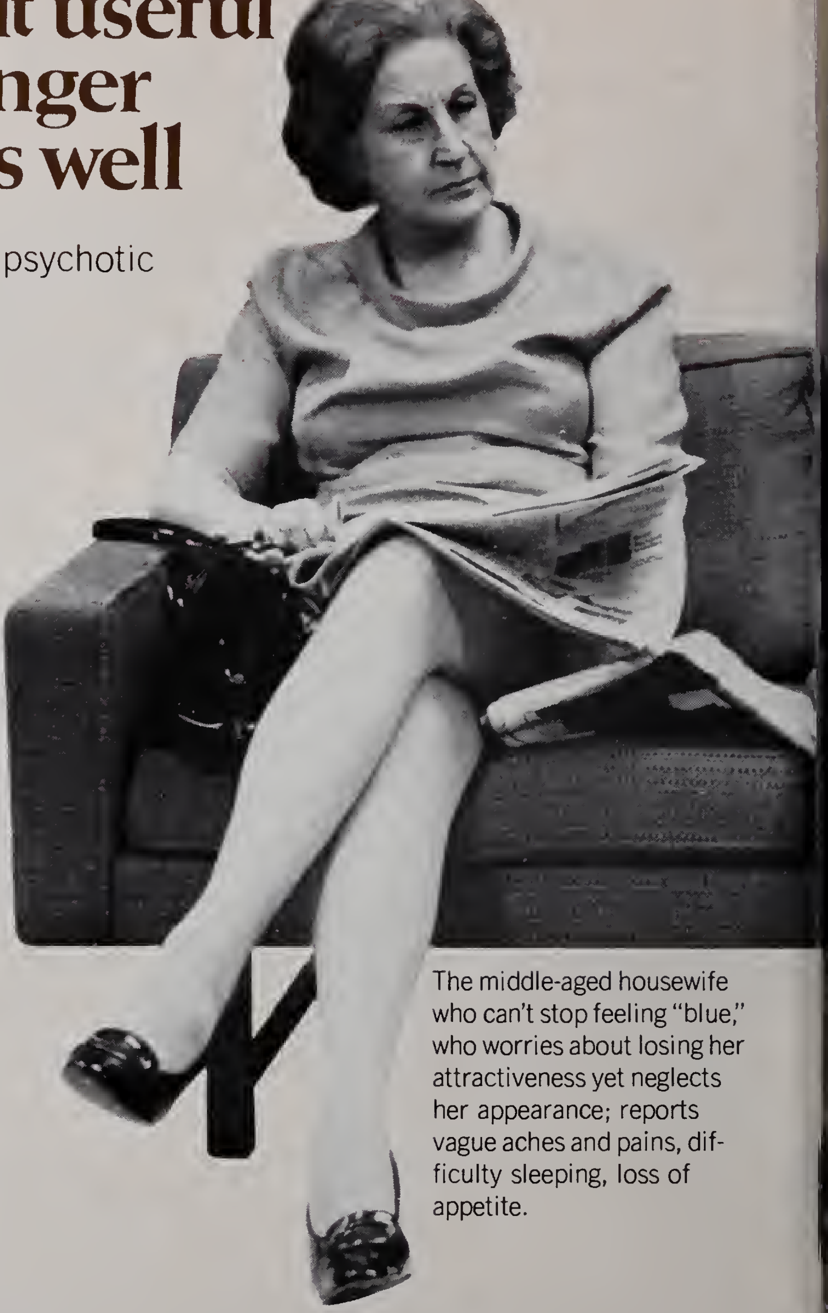

The middle-aged housewife who can't stop feeling "blue," who worries about losing her attractiveness yet neglects her appearance; reports vague aches and pains, difficulty sleeping, loss of appetite.

INDICATIONS: Useful in the management of depression, both acute (reactive) and chronic; particularly useful in the less severe depressions and where the depression is accompanied by anxiety, insomnia, agitation, or rumination; also useful for management of depression and associated anxiety accompanying or related to organic illnesses.

CONTRAINDICATIONS: Benactyzine hydrochloride: Glaucoma and previous allergic or idiosyncratic reactions to benactyzine hydrochloride or related compounds. **Meprobamate**: Acute intermittent porphyria and allergic or idiosyncratic reactions to meprobamate or related compounds such as carisoprodol, mebutamate, tybamate, carbromal.

WARNINGS: The following information on meprobamate pertains to 'Deprol' (meprobamate + benactyzine hydrochloride): **Meprobamate:** *Drug Dependence:* Physical and psychological dependence and abuse have occurred. Chronic intoxication, from prolonged use and usually greater than recommended doses, leads to ataxia, slurred speech, vertigo. Carefully supervise dose and amounts prescribed, and avoid prolonged use, especially in alcoholics and addiction-prone persons. Sudden withdrawal after prolonged and excessive use may precipitate recurrence of pre-existing symptoms (e.g., anxiety, anorexia, insomnia) or withdrawal reactions (e.g., vomiting, ataxia, tremors, muscle twitching, confusional states, hallucinosis; rarely convulsive seizures, more likely in persons with CNS damage or pre-existent or latent convulsive disorders). Therefore, reduce dosage gradually (1-2 weeks) or substitute a short-acting barbiturate, then gradually withdraw. *Potentially Hazardous Tasks:* Driving a motor vehicle or operating machinery. *Additive Effects:* Possible additive effects between meprobamate, alcohol, and other CNS depressants or psychotropic drugs. *Pregnancy and Lactation:* Safe use not established; weigh potential benefits against potential hazards in pregnancy, nursing mothers, or women of childbearing potential. Animal data at five times the maximum recommended human dose show reduction in litter size due to resorption. Meprobamate appears in umbilical cord blood at or near maternal plasma levels, and in breast milk at levels 2-4 times that of maternal plasma. *Children Under Six:* Drug not recommended.

PRECAUTIONS: **Meprobamate**: To avoid oversedation, use lowest effective dose, particularly in elderly and/or debilitated patients. Consider possibility of suicide attempts; dispense least amount of drug feasible at any one time. To avoid excess accumulation, use caution in patients with compromised liver or kidney function. Meprobamate may precipitate seizures in epileptics.

ADVERSE REACTIONS: Nausea, dry mouth, other g.i. symptoms; syncope; one case each of severe nervousness and loss of power of concentration. The following side effects, which have occurred after administration of its components alone, have either occurred or might occur when the combination is taken. **Benactyzine hydrochloride:** Benactyzine hydrochloride alone, particularly in high dosage, may produce dizziness, thought-blocking, a sense of depersonalization, aggravation of anxiety, or distur-

When mild depression
and associated anxiety
interfere with living

DEPROL®

(meprobamate 400 mg +
benactyzine hydrochloride 1 mg)

The junior executive crushed by his repeated failure to be promoted and anxious about the future; complains to you of listlessness, early-morning awakening.

The young widow whose grief has persisted too long, is pessimistic and fearful about what lies ahead, has lost interest in everything; is preoccupied with vague physical ailments, has crying spells.

nce of sleep patterns, and a subjective feeling of muscle relaxation.
ere may also be anticholinergic effects such as blurred vision, dryness
mouth, or failure of visual accommodation. Other reported side effects
ve included gastric distress, allergic response, ataxia, and euphoria.
eprobamate: *Central Nervous System:* Drowsiness, ataxia, dizziness,
urred speech, headache, vertigo, weakness, paresthesias, impairment of
sual accommodation, euphoria, overstimulation, paradoxical excite-
ent, fast EEG activity. *Gastrointestinal:* Nausea, vomiting, diarrhea. *Car-*
ovascular: Palpitations, tachycardia, various forms of arrhythmia, tran-
ent ECG changes, syncope; also, hypotensive crises (including one fatal
se). *Allergic or Idiosyncratic:* Usually after 1-4 doses. Milder reactions:
chy, urticarial, or erythematous maculopapular rash (generalized or
nfined to groin). Others: leukopenia, acute nonthrombocytopenic pur-
ura, petechiae, ecchymoses, eosinophilia, peripheral edema, adenopa-
y, fever, fixed drug eruption with cross reaction to carisoprodol, and
oss sensitivity between meprobamate/mebutamate and meprobamate/
rbromal. More severe, rare hypersensitivity: hyperpyrexia, chills, angio-
urotic edema, bronchospasm, oliguria, anuria, anaphylaxis, erythema
ultiforme, exfoliative dermatitis, stomatitis, proctitis, Stevens-Johnson
ndrome; bullous dermatitis (one fatal case after meprobamate plus
ednisolone). Stop drug, treat symptomatically (e.g., possible use of
inephrine, antihistamines, and in severe cases corticosteroids). *Hema-*
logic: Agranulocytosis and aplastic anemia (rarely fatal), but no causal relationship established. Rarely, thrombocytopenic purpura. *Other:* Exacerbation of porphyric symptoms.

USUAL ADULT DOSAGE: One tablet three or four times daily, which may be increased gradually to six tablets daily and gradually reduced to maintenance levels upon establishment of relief. Doses above six tablets daily are not recommended.

OVERDOSAGE: Overdosage of 'Deprol' (meprobamate + benactyzine hydrochloride) has not differed substantially from meprobamate overdosage: **Meprobamate**: Suicidal attempts with meprobamate, alone or with alcohol or other CNS depressants or psychotropic drugs, have produced drowsiness, lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse, and death. Empty stomach, treat symptomatically; cautiously give respiratory assistance, CNS stimulants, pressor agents as needed. Meprobamate is metabolized in the liver and excreted by the kidney. Diuresis and dialysis have been used successfully. Carefully monitor urinary output; avoid overhydration; observe for possible relapse due to incomplete gastric emptying and delayed absorption. REV 10/71

Before prescribing, consult package circular or latest PDR information.

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Rapid onset of action for the up-tight back in pain
(including intervertebral disc)

Indications: For symptomatic relief in conditions characterized by skeletal muscle spasm and mild to moderate pain.

Contraindications: Acute intermittent porphyria and allergic or idiosyncratic reactions to carisoprodol or related compounds such as meprobamate, mebutamate, tybamate.

Warnings: *Idiosyncratic Reactions:* Rarely, first dose has been followed by extreme weakness, transient quadriplegia, dizziness, ataxia, temporary vision loss, diplopia, mydriasis, dysarthria, agitation, euphoria, confusion, disorientation. Symptoms usually subside during the next several hours. Supportive and symptomatic therapy, including hospitalization, may be necessary. *Pregnancy and Lactation:* Safe use not established; weigh potential benefits against potential hazards in pregnancy, nursing mothers, or women of childbearing potential. *Children Under Five:* Drug not recommended. *Potentially Hazardous Tasks:* Driving a motor vehicle or operating machinery. *Additive Effects:* Possible additive effects between carisoprodol, alcohol, and other CNS depressants or psychotropic drugs. *Drug Dependence:* Use cautiously in addiction-prone patients.

Precautions: To avoid excess accumulation, use caution in patients with compromised liver or kidney function.

Adverse Reactions: *Central Nervous System:* Drowsiness, dizziness, vertigo, ataxia, tremor, agitation, irritability, headache, depressive reactions, syncope, insomnia. *Allergic or Idiosyncratic:* Usually seen after 1-4 doses in patients not previously exposed e.g., rash, erythema multiforme, pruritus, eosinophilia, fixed drug

Soma® 350
(carisoprodol)

Helps to...
- Relax muscle spasm
- Relieve associated mild-to-moderate pain
- Reduce stiffness

Helps give the patient...
- An opportunity to resume daily activities quickly

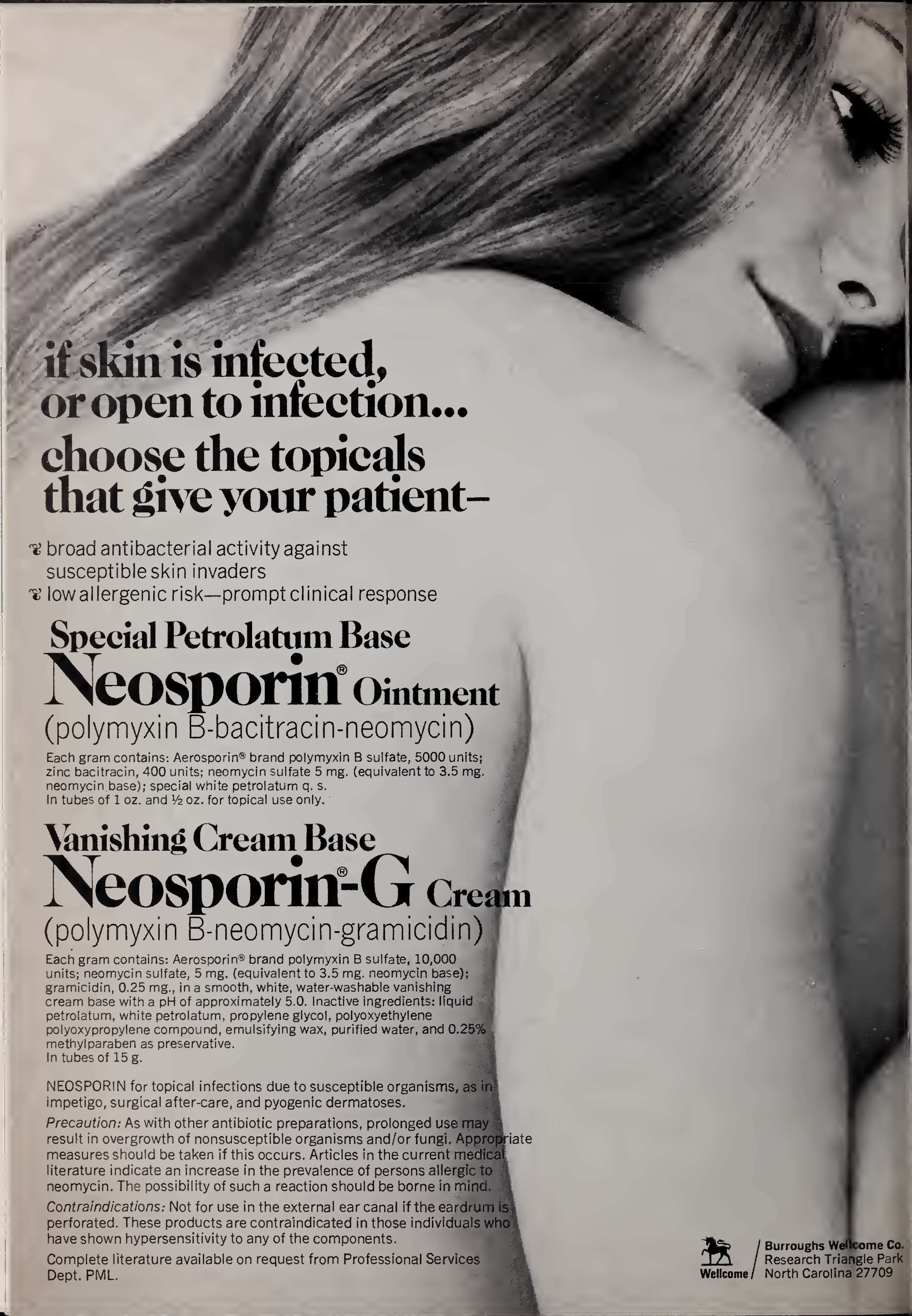
Simple, economical dosage schedule...
- Usual adult dosage: one 350 mg tablet q.i.d.

eruption with cross reaction to meprobamate. More severe manifestations: asthma, fever, weakness, dizziness, angioneurotic edema, smarting eyes, hypotension, anaphylactoid shock. Stop drug, treat symptomatically (e.g., possible use of epinephrine, antihistamines, and in severe cases corticosteroids). *Cardiovascular:* Tachycardia, postural hypotension, facial flushing. *Gastrointestinal:* Nausea, vomiting, hiccup, epigastric distress. *Hematologic:* Leukopenia and pancytopenia (on carisoprodol plus other drugs).

Usual Adult Dosage: One 350 mg tablet three times daily and at bedtime.

Overdosage: Has produced stupor, coma, shock, respiratory depression, and, very rarely, death. Overdosage of carisoprodol plus alcohol or other CNS depressants or psychotropic drugs can be additive. Empty stomach, treat symptomatically; cautiously give respiratory assistance, CNS stimulants, pressor agents as needed. Carisoprodol is metabolized in the liver and excreted by the kidney. Diuresis and dialysis have been used successfully with related drug meprobamate. Carefully monitor urinary output; avoid overhydration; observe for possible relapse due to incomplete gastric emptying and delayed absorption. REV. 10/71

WALLACE PHARMACEUTICALS /Cranbury, N.J. 08512



if skin is infected, or open to infection...

choose the topicals that give your patient–

- broad antibacterial activity against susceptible skin invaders
- low allergenic risk—prompt clinical response

Special Petrolatum Base

Neosporin® Ointment

(polymyxin B-bacitracin-neomycin)

Each gram contains: Aerosporin® brand polymyxin B sulfate, 5000 units; zinc bacitracin, 400 units; neomycin sulfate 5 mg. (equivalent to 3.5 mg. neomycin base); special white petrolatum q. s.
In tubes of 1 oz. and ½ oz. for topical use only.

Vanishing Cream Base

Neosporin®-G Cream

(polymyxin B-neomycin-gramicidin)

Each gram contains: Aerosporin® brand polymyxin B sulfate, 10,000 units; neomycin sulfate, 5 mg. (equivalent to 3.5 mg. neomycin base); gramicidin, 0.25 mg., in a smooth, white, water-washable vanishing cream base with a pH of approximately 5.0. Inactive ingredients: liquid petrolatum, white petrolatum, propylene glycol, polyoxyethylene polyoxypropylene compound, emulsifying wax, purified water, and 0.25% methylparaben as preservative.
In tubes of 15 g.

NEOSPORIN for topical infections due to susceptible organisms, as in impetigo, surgical after-care, and pyogenic dermatoses.

Precaution: As with other antibiotic preparations, prolonged use may result in overgrowth of nonsusceptible organisms and/or fungi. Appropriate measures should be taken if this occurs. Articles in the current medical literature indicate an increase in the prevalence of persons allergic to neomycin. The possibility of such a reaction should be borne in mind.

Contraindications: Not for use in the external ear canal if the eardrum is perforated. These products are contraindicated in those individuals who have shown hypersensitivity to any of the components.

Complete literature available on request from Professional Services Dept. PML.

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The National Congress on the Quality of Life will focus on these immutable concerns by concentrating on the first stages of life's continuum—maternal and child health from conception through adolescence and young adulthood.

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Major emphasis will be on developing new programs that will contribute to the richness and depth of life...programs sensitive to the physical, psychological, emotional and social needs of people...programs reflecting a trend toward more multidisciplinary cooperation.

Don't miss this opportunity to participate in the National Congress. Mark your calendar. Complete the form below.

Sponsored by the American Medical Association in cooperation with 20 governmental and volunteer agencies.

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American Medical Association
535 N. Dearborn Street
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Please forward the program for the National Congress oh the Quality of Life to:

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Affiliation ______________________

Address ______________________

City ______________ State ________ Zip Code ________

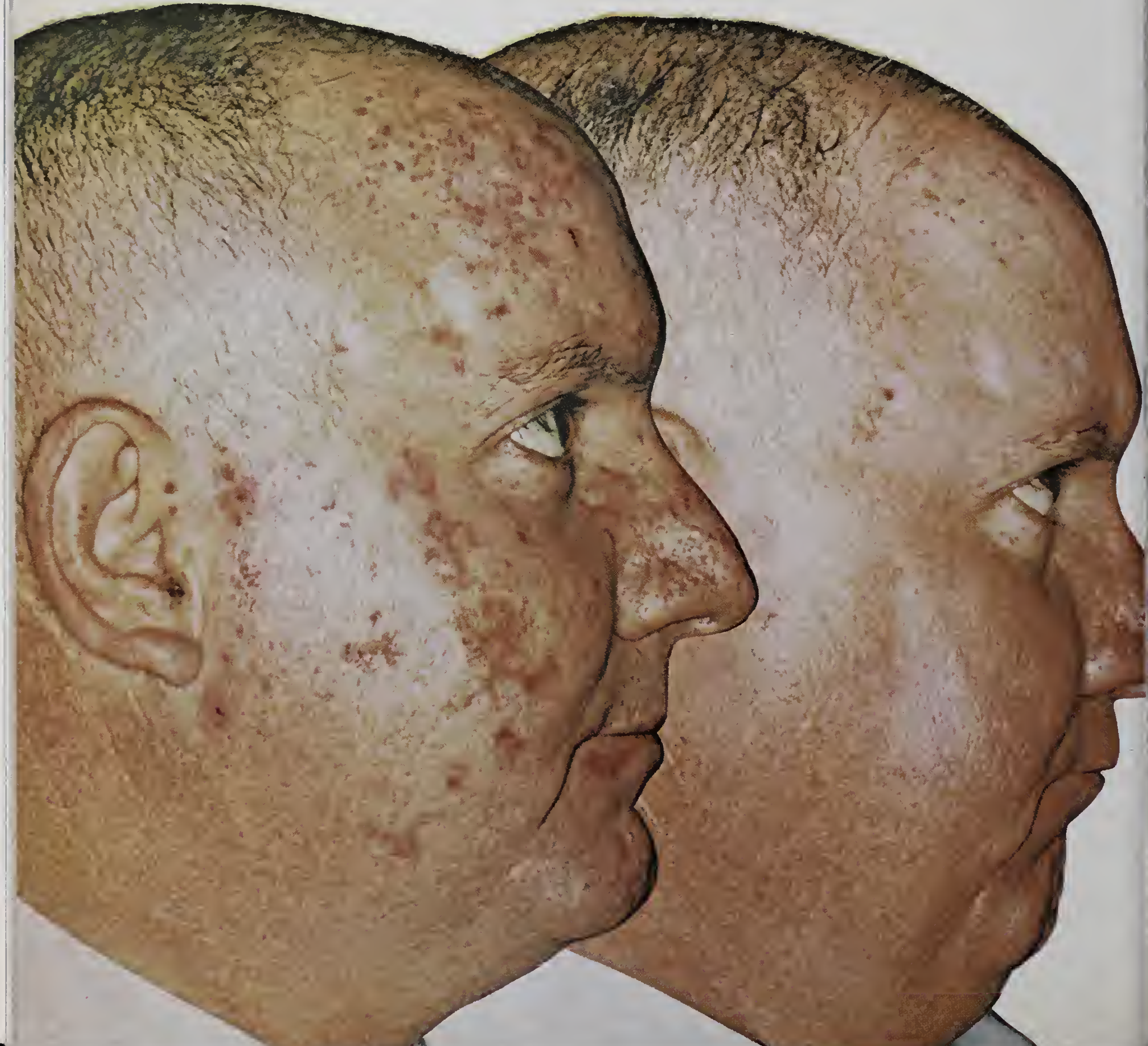
Call it what you will, it may be premalignant...

Before

3/29/67 Before therapy with 5%-FU cream. Patient P. T. shows a moderately severe solar keratotic involvement. Note residual scarring from the previous cryosurgical and electrosurgical procedures on forehead and ridge of nose adjacent to periauricular area.

After

6/12/67 Seven weeks after cessation of therapy. Reactions have subsided. Residual scarring is not seen except for that due to prior surgery. Inflammation has disappeared and face is clear of keratotic lesions.



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Nutley, N.J. 07110

and Efudex® (fluorouracil) 5% cream can resolve it.

all it actinic, solar or senile keratoses,
any regard it as "precancerous."[1,2]

opical fluorouracil, considered by some dermatologists to be a major
dvance in the treatment of multiple solar keratoses,[3,4] offers the physi-
ian a relatively inexpensive alternative to cryosurgery, electrodesic-
ation and cold knife surgery. Of the topical fluorouracils available, only
fudex offers 2% and 5% solution and 5% cream formulations—formula-
ions that have proved effective in the treatment of these mutliple lesions.

sual duration of therapy, 2 to 4 weeks.

tudies showed that with the 2% and 5% Efudex preparations, the usual
uration of therapy was only 2 to 4 weeks.[5] Other studies with topical
uorouracil revealed that when concentrations of less than 2% were
sed, significant numbers of lesions recurred.[6]

reats the lesions you can't see, too.

umerous lesions, not apparent prior to 2% and 5% Efudex therapy,
anifested themselves by definite reactions, while intervening skin
emained relatively unaffected.[5] The early eradication of these subclini-
al lesions (which may otherwise have undergone further progression)
robably accounts for the reduced incidence of future solar keratoses in
atients treated with topical fluorouracil—especially with 5%
oncentrations.[6]

ow to identify solar keratoses.

ypically, the lesion—a flat or slightly elevated brown to red-brown
apule—is dry, rough, adherent and sharply defined. Multiple lesions
re the rule.

redictable therapeutic response.

he response to a typical course of Efudex therapy is usually
haracteristic and predictable. After 3 or 4 days of treatment, erythema
egins to appear in the area of keratoses. This is followed by a moderate
intense inflammatory response, scaling and occasionally moderate
enderness or pain. The height of this response generally occurs two
eeks after the start of therapy and then begins to subside as treatment
stopped. Within two weeks of discontinuing medication, the
nflammation is usually gone. Lesions that do not respond should
e biopsied.

eferences: 1. Allen, A. C.: *The Skin, A Clinicopathological Treatise*, ed. 2, New York,
rune & Stratton, 1967, p. 842. 2. Dillaha, C. J.; Jansen, G. T., and Honeycutt, W. M.:
Treatment of Actinic Keratoses with Topical Fluorouracil," in Waisman, M. (ed.):
harmaceutical Therapeutics in Dermatology, Springfield, Ill., Charles C Thomas, 1968,
. 92. 3. Belisario, J. C.: *Cutis*, 6:293, 1970. 4. Sams, W. M.: *Arch. Derm.*, 97:14, 1968.
. Data on file, Hoffmann-La Roche Inc., Nutley, New Jersey. 6. Williams, A. C., and
lein, E.: *Cancer*, 25:450, 1970.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Multiple actinic or solar keratoses.

Contraindications: Patients with known hypersensitivity to any of its components.

Warnings: If occlusive dressing used, may increase inflammatory reactions in adjacent normal skin. Avoid prolonged exposure to ultraviolet rays. Safe use in pregnancy not established.

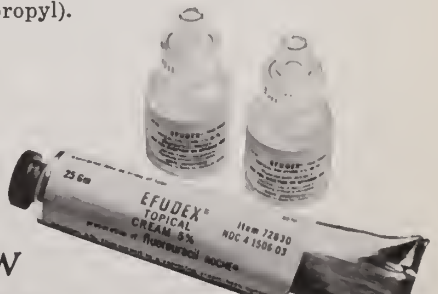
Precautions: If applied with fingers, wash hands immediately. Apply with care near eyes, nose and mouth. Lesions failing to respond or recurring should be biopsied.

Adverse Reactions: Local—pain, pruritus, hyperpigmentation and burning at application site most frequent; also dermatitis, scarring, soreness and tenderness. Also reported—insomnia, stomatitis, suppuration, scaling, swelling, irritability, medicinal taste, photosensitivity, lacrimation, leukocytosis, thrombocytopenia, toxic granulation and eosinophilia.

Dosage and Administration: Apply sufficient quantity to cover lesion twice daily with nonmetal applicator or suitable glove. Usual duration of therapy is 2 to 4 weeks.

How Supplied: Solution, 10-ml drop dispensers—containing 2% or 5% fluorouracil on a weight/weight basis, compounded with propylene glycol, tris(hydroxymethyl)aminomethane, hydroxypropyl cellulose, parabens (methyl and propyl) and disodium edetate.

Cream, 25-Gm tubes—containing 5% fluorouracil in a vanishing cream base consisting of white petrolatum, stearyl alcohol, propylene glycol, polysorbate 60 and parabens (methyl and propyl).



now
Efudex®
(fluorouracil)
cream/solution

The creative use of free time. With no television.

The staff do not like to think of themselves as just another institution for special education. They had rather work with behavior problems as such. But it is difficult to create a mentally healthy, goal oriented adult in our society if that adult cannot read and write and do at least basic arithmetic. So individualized remedial teaching is an integral part of the Center's activity. The learning periods must be short, and successes rewarded frequently in the beginning, but remarkable progress is often made and before long the child may well be back in the public schools.

Psychotherapy and one-to-one counseling relationships are also provided the child, and to the parents also. As many as one-fourth of the parents may be in group therapy at any given time, elucidating their role in the child's mental health as well as in their own.

Occupational therapy, manual arts therapy and recreational therapy will be integral parts of Michael's program, and a psychiatrist will come in once a week to check on any medication schedule he may have been placed on elsewhere, and scan him for any obvious medical needs. A nurse will be available at all times.

Michael's program will be on a five-day-a-week basis between the hours of 8:30 a.m. and 2:30 p.m. He will be expected to get to the Center on his own and be free from any physical or medical complications that must be treated elsewhere.

How does the Center shape up as a professional unit? The staff is a multi-disciplinary group consisting of a clinical child psychologist, 2 special education teachers, 2 social workers, 4 counselors, 3 recreational therapists, an occupational therapist, a nurse, a part-time psychiatrist, a secretary and a director. Students from area colleges and universities also serve internships at the Center. A better than 1:3 staff-patient ratio assures the individualized attention required by children with behavioral and emotional disorders.

The setting may be less than ideal: Situated on the D. C. General Hospital grounds across the street from the Morgue; housed in a converted hospital chapel with a closed ward of women alcoholics upstairs and a multitude of ancient, grey tombstones looming up from the cemetery outside their "quiet room" windows. And everyone feels that there should be 400 children in the Center instead of 40. And the Mental Health Administration feels that when the public wants it bad enough it will no doubt come about.

Meanwhile, Michael will be hugging that old shaggy dog in about 3 weeks. Give him another four months and he will be reading stories to him out of a fifth grade teacher. Another 4 months and he will probably have the biggest part of his childhood back again.

ESSEX C. NOEL, III, M.D.
Director,
D.C. Mental Health Administration

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d.c. HEALTH NOTES

Family Health Center Aide Program

The Community Health and Hospitals Services Administration of the Department of Human Resources has successfully implemented another program—Family-Health Center Aides—which will be of much value to the people of the District and to the Health Centers to which the Aides have been assigned.

This program follows closely another innovative program—the Pediatric Nurse Associate (Practitioner) program—in which we more effectively utilize the skills of the nurse in providing health services for infants and children through specialized training in total patient care.

It was my pleasure and privilege to be the guest speaker at the Ceremony and Reception for the Aides at the Health Center for Mothers and Children, 4130 Hunt Place, N. E. Framed certificates, noting successful completion of the course, were presented to Ophelia Dempsey, Geraldine Fleming, Celeste King, Versey McLaughlin, Katherine Robinson, Little Mae Taylor, Ellen Johnson, Mary Keenon, Geraldine Hailey and Naomi Turner.

The capacity audience roundly applauded the graduates and signified solid support behind the Family-Health Center Aides Program, which is under the direction of Dr. Roselyn P. Epps, Chief of the Bureau of Maternal and Child Health.

The Family-Health Center Aides are a group of subprofessional workers, and are residents of the Northeast area of the District of Columbia, who were trained for their duties through the Public Service Careers Program under the Children and Youth Project #631.

Selected from the 40 persons applying for the program, the 10 Aides had the following qualifications:

1. They are full-time residents in the Project area;
2. They have their own children or experience relating to and working with children;
3. They are alert, stable and empathetic.

The program was launched on June 28, 1971 with 10 aides enrolled. The curriculum included five weeks of didactic instruction, three days of which was at Hannah Harrison School and two days on field trips and in the Health Centers. Instructors in social service, nursing and nutrition were recruited and a nurse from the staff was detailed to act as supervisor and coordinator.

On-the-job training was continued following the initial five weeks. The Aides have completed six months of training and are currently working with all professional disciplines involved in the Child Activity Program. As residents of the community, the Aides have a vested interest in the patients served by the Centers, and they are well accepted as our health representatives.

Functioning both in the community and in the Health Centers, the Aides are truly unique and unlike most other aide programs or sub-professional groups. The Aides not only make home visits, but they also have assignments within the Health Centers working with the physicians, dentists, nurses, social workers, nutritionists, audiological and speech pathologist and other staff. Their acceptance by the clinic staff and the community has been more than enthusiastic.

RAYMOND L. STANDARD, M.D., M.P.H.
Director of Public Health and
Administrator, Community Health and
Hospital Administration
Department of Human Resources

NEW MEMBERS

Active

NORMAN A. ARMSTRONG, M.D. Office: 850 Delaware Avenue, S.W. Born in Frederiksted, St. Croix, U.S. Virgin Islands. Medical degree: Howard University, 1970. Specialty: General Medicine.

SYLVESTER C. BOOKER, M.D. Office: 607 4th Street, S.W. Born in Richmond, Virginia. Medical degree: Howard University, 1964. Specialty: Obstetrics-Gynecology.

ROSWELL G. DANIELS, M.D. Office: 719 13th Street, N.W. Born in Watertown, New York. Medical degree: University of Rochester, 1947. Specialty: Occupational Medicine.

HALAPPA G. HAKKAL, M.D. Office: D.C. General Hospital. Born in Yelodahalli, Mysore, India. Medical degree: Mysore Medical College, 1965. Specialty: Radiology.

MOHAMMED HAZIQ, M.D. Office: 9401 Indian Head Highway. Born in Bombay, India. Medical degree: Dow Medical College, 1963. Specialty: Urology.

FRANCIS G. LAPIANA, M.D. Office: Armed Forces Institute of Pathology. Born in Detroit, Michigan. Medical degree: George Washington University, 1962. Specialty: Ophthalmology.

DEVRA MARCUS, M.D. Office: 3308 14th Street, N.W. Born in Bronx, New York. Medical degree: Stanford University, 1966. Specialty: Internal Medicine.

PHILLIP O. PERIMAN, M.D. Office: George Washington University Clinic. Born in Memphis, Texas. Medical degree: Washington University, 1965. Specialty: Internal Medicine.

DIETER SCHELLINGER, M.D. Office: Georgetown University Hospital. Born in Buchau, Germany. Medical degree: University of Dusseldorf, 1962. Specialty: Radiology.

Associate

LESLIE CHARLES, M.D. Office: Freedmen's Hospital. Born in Trinidad, West Indies. Medical degree: Meharry Medical College, 1964. Specialty: Obstetrics-Gynecology.

JOEL COHEN, M.D. Office: Walter Reed Hospital. Born in Bronx, New York. Medical degree: New York Medical College, 1967. Specialty: Psychiatry.

MARIANO GARCIA, M.D. Office: 10401 Old Georgetown Road. Born in Cuba. Medical degree: University of Havana, 1960. Specialty: Endocrinology.

JAMES JOHN LINN, M.D. Offices: Washington Hospital Center and Warren Memorial Hospital, Front Royal, Virginia. Born in Tampa, Florida. Medical degree: Tulane Medical School, 1966. Specialty: Pathology.

WALLACE MENDELSON, M.D. Office: St. Elizabeths Hospital. Born in Eagle Pass, Texas. Medical degree: Washington University, 1969. Specialty: General Practice and Psychiatry.

Changes in Membership

Transfers

Active to Special: Howard H. Strine, M.D., James E. Wissler, M.D.

Active to Associate: Marvin L. Adland, M.D.

Resignations

Active: Eloise W. Kailin, M.D., Alfred A. Leszczynski, M.D.

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MEDICO-LEGAL REPORT

The District of Columbia Professional Corporation Act

On December 10, 1971, the District of Columbia Professional Corporation Act (the "Act") became law, enabling certain professional individuals and firms in the District of Columbia to incorporate. The Act covers physicians, dentists, optometrists, podiatrists and others who are authorized to render professional services to the public in the District of Columbia. While the advantages of incorporating a medical practice can be quite substantial, and have been well publicized, each practitioner must also carefully evaluate the requirements of the Act, as well as the burdens, disadvantages and risks of incorporating.

Set forth below is a description of the important features of the Act, as well as a brief summary of some relevant factors which must be taken into account in deciding whether to incorporate a professional practice.

Requirements of the Act

A professional corporation under the laws of the District of Columbia (a "P.C.") may render professional services through licensed individuals, charge and collect for services, and compensate those who render such services. A P.C. may also perform ancillary services through non-licensed personnel. A P.C. is permitted to engage in a limited amount of investment-type business activities; however, it is generally more advantageous for tax reasons that these activities be undertaken by a pension and/or profit-sharing plan adopted by the P.C., as more fully discussed below.

The following formalities must be met by a P.C. under the Act: (1) The articles of incorporation of the P.C. must meet the requirements of the District of Columbia Business Corporation Act as well as set forth other specific information; (2) a P.C. must have one or more directors; (3) generally, only a licensed professional may serve as a shareholder, officer or director of the P.C.; (4) a P.C. must render services and exercise its powers under a name which includes the words "professional corporation", the abbreviation "P.C." or the word "chartered"; and (5) no shareholder may enter into any arrangement with anyone other than another shareholder of the P.C. under which he relinquishes his right to vote as a shareholder.

The importance of observing the required formalities in every possible way cannot be overemphasized. It is essential that professionals who have formed a P.C. do not conduct their practices as individuals; otherwise, there is the risk that the Internal Revenue Service will disregard the P.C. and tax its income directly to the professional who earned it, which would have disastrous income tax consequences.

The professional and confidential relationship between the physician and his patient is preserved under the Act. However, a professional is liable only for malpractice committed by him or an individual rendering professional service under his supervision and control, and not for the malpractice of other licensed professionals employed by the P.C. By comparison, where a professional practice is conducted in partnership form, each professional is liable for the malpractice of each other professional who is his partner. This is a major non-tax advantage of a P.C., although in many instances this problem can largely be solved by each professional obtaining coverage under a liability insurance policy. With respect to malpractice or negligence, the P.C. is liable up to the full value of its assets for the malpractice of the professionals or others which it employs.

There are specific provisions in the Act relating to the transfer of shares of stock in the P.C. Sales and other transfers of shares are exempt from regulation under Federal or local securities laws. Shares may be sold to other professionals; however, by provision in the corporate documents, shareholder approval may be required for any such sale or transfer of P.C. stock. Also, any stock validly sold or transferred will continue to be subject to the requirements of the Act, and a legend must be imprinted on the face of the stock certificate to this effect. Other transfers of stock permitted under the Act include (1) reacquisition by the P.C., (2) transfers to creditors for individual debts, and (3) transfers upon the death, legal incompetence, retirement, expulsion or disqualification to practice of a professional; however, in the latter two cases, the P.C. has the right to redeem the shares of stock from the outsider within a specified time limit.

Corporations licensed in other jurisdictions may be authorized to perform professional services in the District of Columbia if they meet certain requirements of the Act, and if such other jurisdiction reciprocally authorizes P.C.'s to render professional services in such other jurisdiction.

Finally, while the District of Columbia corporate income tax is applicable to P.C.'s, the unincorporated business tax generally applicable to trades or businesses which are permitted to incorporate in the District of Columbia will not be applicable to professionals whose sole right to incorporate is under the Act and who do not choose to incorporate.

The Decision to Incorporate

The decision of whether to incorporate requires an evaluation of the tax and non-tax advantages and disadvantages, and this analysis will vary from case to case.

The most important tax benefit to be derived from incorporating a professional practice is that the P.C. can make substantial contributions of a percentage of the professional's (and other employees') salary under a qualified pension and/or profit-sharing plan. Such contributions are tax-deductible by the P.C. and exempt from tax to an employee until distributed to him. In addition, the contributions are made to a tax-exempt trust so that income and appreciation of the funds accrue tax-free, until distributed. The P.C. may contribute up to 25 per cent or more of the professional's (and other employees') salary.

The threshold question that must be answered in deciding whether to incorporate is whether the professional, on the basis of his standard of living, can afford the reduction of spendable income resulting from a substantial annual contribution by the P.C. to the pension and/or profit-sharing plan to justify the initial morass of paper work and the continuing obligations of maintaining a P.C. (such as maintaining minute books and records, holding annual meetings, filing annual reports and complying with the other required formalities described above), to say nothing of the legal and accounting fees involved and the outlay of additional compensation to nonprofessional employees who are required to be covered under the pension and/or profit-sharing plan.

Other fringe tax benefits are as follows:

(1) Payments of premiums for group-term life insurance for the benefit of employees are tax-deductible by the P.C. and, to the extent of up to $50,000 of coverage, are nontaxable to the employee.

(2) Reimbursements by the P.C. for medical and dental expenses incurred by employees, including the cost of health insurance premiums, are deductible by the P.C. and tax-free to employees to whom they are made.

(3) Payments under a wage-continuation plan in the event of an employee's sickness or disability are deductible by the P.C. and, within specified limits, tax-free to the employee.

(4) Death benefits are deductible by the P.C. and are exempt from tax to a deceased employee's beneficiary to the extent of $5,000, plus an additional amount may be tax-exempt in the case of certain payments to the widow of a deceased employee.

As to tax risks involved, any income accumulated by the P.C. will be subject to double tax: a corporate tax when earned, and a tax to the shareholders upon the subsequent payment of dividends and/or a liquidating distribution. If the P.C. attempts to defer the second tax by refraining from paying dividends to the shareholders, there is the possibility of attack by the Internal Revenue Service on the grounds that the P.C. is a personal holding company and/or is unreasonably accumulating income, each of which, if upheld, will result in substantial penalty taxes. If the P.C. attempts to avoid this problem by paying out all of its income in the form of salaries, bonuses, fringe benefits, and contributions to a pension and/or profit-sharing plan, there is the risk that the Service will attack the compensation paid to a particular employee as excessive; and, if so, the P.C. will lose its deduction for payments which are considered excess compensation. Furthermore, such excess compensation would lose its status as "earned income" of an employee subject to the maximum tax at a 50 per cent rate for taxable years beginning after December 31, 1971. While it may seem unlikely to a professional that his compensation can be considered excessive if he essentially produced the very income paid to him as compensation, the Service may nevertheless rely upon a standard of comparability to other professionals or other corporate executives in making its determination.

There are a number of non-tax advantages of a professional corporation. In addition to the freedom from responsibility of malpractice of a fellow practitioner as mentioned above, the P.C. and its patients' files have a perpetual life, thereby avoiding the problems often faced by a partnership upon the death of a professional partner. Furthermore, transferability of stock of a corporation by purchase or redemption may be simpler and freer under corporate form by comparison to the complexities of transfers of partnership interests.

Needless to say, the judgment can only be made after a thorough analysis of all of the facts involved. However, the District of Columbia Professional Corporation Act has been long awaited by licensed professionals in the District of Columbia to give them the opportunity to be treated comparably to other business corporations which have long enjoyed the benefits of operating in corporate form.

MICHAEL R. FLYER, J. D.

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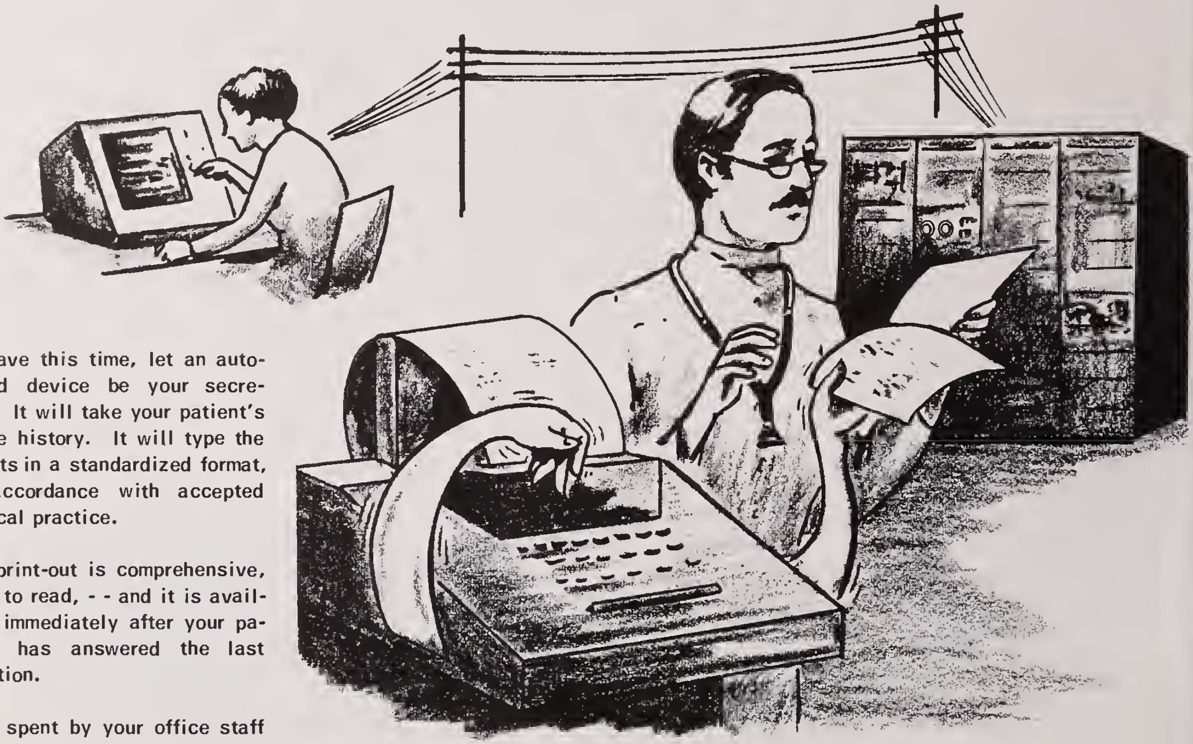

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book REVIEWS

"Eye Symptoms in Brain Tumors", 2nd ed. ALFRED HUBER, M.D. Edited and Translated by Frederick Blodi, M.D. Price $32.50 Pp. 376 with 233 illustrations. St. Louis: C.V. Mosby, 1971.

Those of us who own the first edition reporting Dr. Alfred Huber's experience in analyzing ocular symptoms and syndromes seen in association with brain tumors, welcome this second edition.

It is based on the personal experience of the author in evaluating 2500 cases of brain tumors on record in the Neurosurgical Clinic at the University of Zurich. Dr. Huber has had the rare privilege of serving as consultant in ophthalmology to the neurosurgical unit where he has had the intimate association of such giants of neurosurgery as Professors H. Krayenbühl and G. M. Yasargil.

Frederick C. Blodi, Professor and Chairman of the Department of Ophthalmology of the University of Iowa College of Medicine, has faithfully translated and skillfully edited the original German text in a lucid style. He has avoided the awkward cumbersome sentence structure often seen in translated manuscripts.

In the chapter on methods of neuro-ophthalmologic examination, the author has up-dated the original text introducing the reader to static perimetry in the study of visual fields, electromyography in evaluation of muscle palsies and fluorescein angiography in observations of the optic disc under normal and pathologic conditions.

The author presents a most informative chapter dedicated to the general symptoms of increased intracranial pressure. It should be read by all interested in the differential diagnosis of papilledema and includes methods of diagnosing the early incipient changes. The "clivus ridge syndrome" (compression of the oculomotor nerve by protruding brain substance at the tentorial notch), is included in this chapter and represents one of the best discussions of this subject ever read by the reviewer.

A logical and orderly sequence then follows, presenting ocular symptoms of brain tumors from a topographical viewpoint in Chapter 3 and from a histologic viewpoint in Chapter 4. A special section deals with metastatic tumors to the brain. The last chapter deals with tumor-like changes, presenting ocular symptoms due to pseudotumor cerebri, subdural hematomas, brain abscesses and aneurysms.

The author has compiled a very adequate bibliography in which the classical articles have been collected and categorized under various subject headings. This is invaluable and affords an easy up-to-date review of the world literature.

This is an excellent book and should be owned by anyone interested in Neuro-ophthalmology.

MELVIN G. ALPER, M.D.

Immunology. JOSEPH A. BELLANTI, M. D. Price, $15.75. Pp. 584 with illustrations. Philadelphia: W. B. Saunders Company, 1971.

Dr. Bellanti, as both a Pediatrician and Immunologist, has recognized the necessity for change in the approach to teaching Immunology as a clinical as well as a laboratory science in the modern medical curriculum. To this end, he has written a well integrated, easily readable text which presents both the facts of basic Immunology and the use of these facts in an approach to a clinical problem. The organization of this textbook presents the subject in three broad sections. The first presents an historical introduction, followed by chapters dealing with the principals of Immunobiology, Immunogenetics and Immunochemistry. This is followed by a section dealing with the mechanisms of immune response dealing with inflammation, tissue injury, infectious agents and the immunologic aspects of Neoplasia. The latter subject is one which is well summarized in this text and usually is not adequately discussed in Immunology textbooks of general interest. The third section deals with the usual subjects of clinical Immunology and presents them in a well illustrated and easily understood format. The final chapter entitled "Diagnostic applications of Immunology" provides a brief but thorough outline of the methods of assessing immunologic function. Dr. Bellanti continues his clinical applications of Immunology through a series of appendices which delineate the approach to a variety of common problems in patients with immunologic disease, and the application of Immunology to other fields of medicine. In summary, this text approaches the subject of Immunology in a somewhat refreshing and clinically oriented manner which should make this a very desirable textbook for many practicing physicians as well as students and house officers.

NORMAN C. KRAMER, M.D.

"Ophthalmic Ultrasound: An International Symposium," KURT A. GITTER, M.D., ARTHUR H. KEENEY, M.D., D.Sc., LOV K. SARIN, M.D., AND DAVID MEYER, M.D. Price, $29.50. Pp. 374 with 623 illustrations on 296 figures. St. Louis: C. V. Mosby, 1969.

Seventy scientists from all over the world made contributions to this book on ultrasound. Besides physicians, the participants included engineers, biophysicists and electronic technologists.

The first section of this book describes basic physics of ultrasound, engineering concepts of acoustic waves and various types of generators and receivers. Although very technical, there are very good descriptions and illustrations in this first section which enable the ophthalmologist to understand the various modalities that are being used in the ophthalmic ultrasound today.

Experimental studies of the behavior of ultrasound in the eye are described in the section on ophthalmic ultrasound. Time-amplitude ultrasonography (TAU) and scanned intensity modulated ultrasonography (SIMU) are discussed rather thoroughly, giving the advantages and disadvantages of each.

Biometric studies in the third unit of this book deal with the various measurements of the eye by ultrasound with statistical comparisons by other methods. Anatomical and pathological measurements are similarly compared as well as new techniques.

Diagnostic ultrasonography is also extensively discussed. This section is of much greater interest to the clinical ophthalmologist. It relates to ultrasound studies on various pathological changes in the eye and orbit.

Finally, there is a small but very exciting section on ultrasonic therapeutics, highlighted by the well known work of Dr. Kelman, who has used ultrasound in the removal of cataracts.

This book can be recommended to all ophthalmologists and other physicians interested in ultrasound. It is an excellent reference on the latest work in ultrasound while the first chapters furnish the student with the basic principles of ultrasound.

L. EDWARD PERRAUT, M. D.

Management of Juvenile Diabetes Mellitus, 2nd edition. HOWARD S. TRAISMAN. Price, $19.75. Pp. 223 with illustrations. St. Louis: C. V. Mosby Company, 1971.

The author intends this second edition of his monograph primarily for the pediatrician and general practitioner, and for medical students, house officers, and nurses.

Although the author does not neglect the discussion of the physiologic and pathophysiologic principles involved, he does not devote too much attention to these, in keeping with his expressed purpose of providing a concise and practical method of management of juvenile diabetes.

We often find difficulty in answering the question, "Just exactly how do you go about managing your diabetic patient?". Dr. Traisman provides a comprehensive and clear presentation of what to do and how to do it. In addition to thorough discussions of dietary management (including useful food tables), selection and administration of insulins, and instruction of patients and parents, the text provides many useful and practical suggestions from the author's experience. There are charts of sugarless drug preparations, and a helpful outline for fluid and electrolyte therapy for diabetic infants and children.

Additional chapters are contributed by six other authors, including discussions of the infant of the diabetic mother, hypoglycemia, and pancreas transplantation.

This reviewer was especially impressed with the chapter on the psychologic aspects of diabetes. The authors draw on their larger experience, and emphasize that, "If the physician remembers that the child patient with diabetes is first a child, then a diabetic, it may help him to avoid . . .forgetting the possible emotional toll that such a disease is likely to take on the child and his family."

While the $19.75 price tag seems high for this type of publication, the book will prove valuable to the physician or nurse dealing with juvenile diabetes.

SAMUEL D. LOUBE, M.D., F.A.C.P.

Preventive Medicine in World War II, Volume IX, Special Fields. COLONEL ROBERT S. ANDERSON, MC, USA, Editor in Chief. Price, $8.00. Pp. 650 with 84 illustrations, 23 tables, 14 charts and 11 maps. Washington, D.C.: Office of the Surgeon General, Department of the Army, 1969.

This volume, Special Fields, is part of a clinical series included in the administrative or operational series prepared by the Historical Unit, United States Army Medical Department. The contents of this book include chapters dealing with Training, Health Education, Occupational Health and Industrial Medicine, Disabilities Due to Environment and Climatic Factors, Medical Intelligence, Enemy Prisoners of War and Medical Laboratories. Tropical Medicine, Field Training in Malarial Control, Health Sanitation Training for Combat Soldiers and Venereal Disease Control are given wide coverage including Curriculum and Training Aids. The chapter on Health Education is well written with emphasis on the devices used in the control of venereal diseases. Occupational Health and Industrial Disease assumed more importance as the United States Army became more deeply involved with industrial production. There resulted greater responsibility of the Medical Department for the medical care of civilian employees, personal hygiene; and, the effects of heat and cold trauma to personnel.

As was the case in other branches of the services, World War II being global, presented problems that were different in some respects than were heretofore met. Consequently, especially in the case of Medical Intelligence, one had few guidelines to work with, and because of the enormous situation and wide and diverse problems, questions were met for the first time by groups who studied and resolved them as the war progressed.

The chapter, Enemy Prisoners of War, written by Brigadier General Stanhope Bayne-Jones was excellently presented. Reference to the various authoratative international agreements such as the Geneva Convention of 1929, the Red Cross Convention, and the Prisoner of War Convention were somewhat limited and fell short of the global scope of operation. New concepts and regulations had to be formulated. The two theaters of operation considered were the European Theater and the Pacific Theater. Within each area there were many differences as to the psychological attitude of the prisoners, and the aspects as to the conception and treatment of diseases. It was noted that few Japanese were taken alive, and only those who were greatly malnourished and severely wounded were taken as prisoners. On the other hand, when considering the prisoners in the European theater, many were turned over in such numbers that the facilities were strained to adequately take care of them. As a result feeding and hygienic sanitary measures had to be carried out to prevent epidemiological disease outbreaks among them. Newly encountered diseases had to be diagnosed in both the European and Pacific Theaters.

The history of the Army Medical Laboratories were important because one finds the origin of some of the country's major achievements in clinical research and public health laboratory medicine. The variety of laboratory tests required contributed to the marked progress in medicine. Training at the school of aviation medicine concentrated on tropical medicine and the effects of trauma.

The Army Medical Museum was considered to be one of the most notable achievements of Surgeon General William A. Hammond. Today, it is a fine institution located along with the

Armed Forces Institute of Pathology at the Walter Reed General Hospital in Washington, D.C. Personnel associated with these institutions not only were dedicated for furthering the education of the military, but also the civilian medical profession.

Medical Field Laboratories were now established throughout the world. The Army Epidemiological Board was a central board to advise the Surgeon General in the control of epidemic and other respiratory diseases. The membership roster included some of the most outstanding medical doctors in the United States. The United States Public Health Service extended assistance to the army preventive medicine program during the National Emergency. The International Health Committee of Rockefeller Foundation (now University), which for years had been actively engaged in the study of yellow fever, typhus, malaria, hookworm disease, rabies and other important infectious diseases, likewise gave able assistance in carrying out of the programs.

The Medical Department of the United States Army accomplished a tremendous program in the increased establishment, the professional expansion, and the effectiveness of the preventive medicine in the laboratory support in the care of personnel suffering from infectious and communicable diseases during and subsequent to World War II.

To supplement the written text, there are many illustrations of installations, of training aids and of pictures taken of prisoners of war as well as laboratories and mobile laboratories throughout the world. Tables consisted of essential statistical data of the prevalence of diseases, reports of casualty rates, and maps prepared by the Cartographic Draftsman of the Historical Unit. The latter includes both the overall theaters of war and the various areas of combat.

The book is of particular value to those interested in both military and military medical history with emphasis on preventive medicine. The contents are well documented through the use of memoranda, reports, and references to the War Department Manual and the subject matter is presented in a very readable manner.

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medical annals of the district of columbia

vol. 41 no. 3 march 1972

Handbook of Poisoning, 7th edition. ROBERT H. DREISBACH, M.D. Price, $6.00. Pp. 515 with illustrations. Los Altos, California: Lange Medical Publications, 1971.

Professor Dreisbach's justly famous little book has reached a 7th edition in only 16 years, attesting not only to its popularity, but to the rapidity of growth of the field. Reviewing it is comparable to reviewing an encyclopedia; nowhere else is such a mass of information on clinical toxicology available in so compact a format. The current edition is enlarged and largely updated, including the references to current literature; a chapter on poison prevention is welcome. Even the inside covers are utilized for ready reference to first aid recommendations.

A few major errors, selected at random, include:

(1) The recommendation for use of activated charcoal as a first aid measure, to be followed by Syrup of Ipecac emesis. This order should be reversed, since a good grade of activated charcoal will absorb all the emetine and render the Ipecac ineffective; indeed, charcoal is best not used as a first aid measure.

(2) Therapeutic recommendations for the relief of nausea and diarrhea are archaic and should be modernized.

(3) Parenteral fluid therapy is not stated in terms of modern usage; also pediatric aspects of fluid therapy are poorly stated.

(4) In the section on rapid detection of poisons in body fluids, an obvious omission is the use of readily available Phenistix (r) for the detection of phenothiazines and salicylate.

(5) A great deal of space (relatively) is devoted to the technic of performing a tracheostomy, while mentioning cricothyroid membrane puncture only in passing.

Despite the above shortcomings, this little book is a goldmine of useful material. It belongs on the desk of the pediatrician and industrial physician, and on the reference shelf of most other practitioners.

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obituaries

James N. Greear, Jr., M.D.
1895-1971

Dr. James N. Greear, Jr., President of the Medical Society of the District of Columbia 1944-1945, died November 9, 1971. He resided in Reno, Nevada.

Born January 29, 1895 in Toms Creek, Virginia, Dr. Greear received his medical degree from the University of Virginia in 1920. He interned at the University of Virginia Hospital and served residencies at the Episcopal Eye, Ear, Nose and Throat Hospital in Washington, D.C. and the Manhattan Eye, Ear and Throat Hospital in New York.

Dr. Greear was Chief Medical Officer at the Army Medical Center for Ophthalmology, Valley Forge General Hospital and Senior Consultant in Ophthalmology, European Theatre of Operations during World War II.

Professor of Opthalmology at Georgetown University School of Medicine from 1928 to 1940, Dr. Greear was also affiliated with the Episcopal Hospital, Emergency Hospital, Children's Hospital, and Washington Medical Center in Washington, D.C. He later held staff appointments at Washoe Medical Center and St. Mary's Hospital in Reno, Nevada.

In addition to his membership in the Medical Society of the District of Columbia, Dr. Greear was a member of the Washoe County Medical Society in Reno, Nevada, the Nevada State Medical Society, the American Ophthalmologic Society, the American Academy of Opthalmology and Otolaryngology, the American College of Surgeons, and the Virginia Medical Society.

He is survived by his wife, Margaret, two daughters, Mrs. Everett Wilson and Mrs. Thomas Gore; two sons, Charles Sperry Greear and James N. Greear, III, and three sisters.

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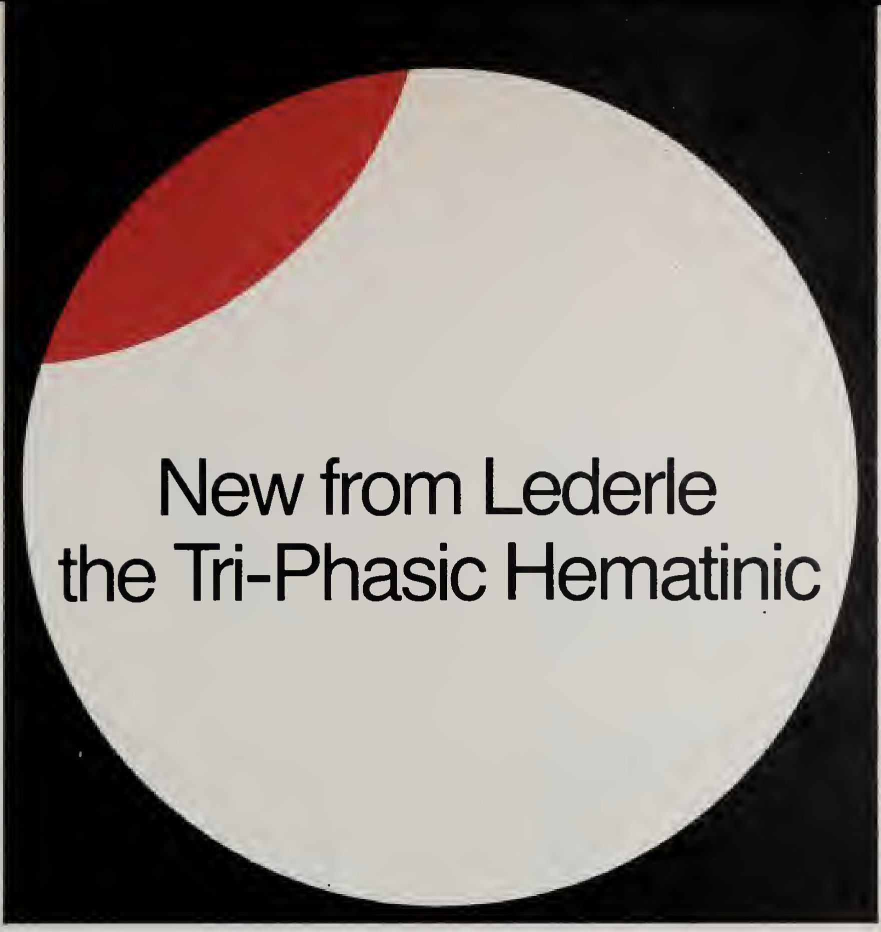

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Runny Nose
THE COLD SYMPTOMS THAT MAKE HIM MISERABLE
ALL IN 'ORNADE':
Drying Agent (isopropamide, as the iodide—2.5 mg.)
Decongestant (phenylpropanolamine HCl—50 mg.)
Antihistamine (chlorpheniramine maleate—8 mg.)
THE INGREDIENTS HE NEEDS FOR PROLONGED RELIEF

Before prescribing, see complete prescribing information in SK&F literature or *PDR*.

Indications: Upper respiratory congestion and hypersecretion associated with: the common cold; acute and chronic sinusitis; vasomotor rhinitis; allergic rhinitis (hay fever, "rose fever," etc.).

Contraindications: Hypersensitivity to any component; concurrent MAO inhibitor therapy; severe hypertension; bronchial asthma; coronary artery disease; stenosing peptic ulcer; pyloroduodenal or bladder neck obstruction. Children under 6.

Warnings: Advise vehicle or machine operators of possible drowsiness. Warn patients of possible additive effects with alcohol and other CNS depressants.

Usage in Pregnancy: In pregnancy, nursing mothers and women who might bear children, weigh potential benefits against hazards. Inhibition of lactation may occur.

Effect on PBI Determination and I^{131} Uptake: Isopropamide iodide may alter PBI test results and will suppress I^{131} uptake. Substitute thyroid tests unaffected by exogenous iodides.

Precautions: Use cautiously in persons with cardiovascular disease, glaucoma, prostatic hypertrophy, hyperthyroidism.

Adverse Reactions: Drowsiness, excessive dryness of nose, throat or mouth; nervousness; or insomnia. Also, nausea, vomiting, epigastric distress, diarrhea, rash, dizziness, weakness, chest tightness, angina pain, abdominal pain, irritability, palpitation, headache, incoordination, tremor, dysuria, difficulty in urination, thrombocytopenia, leukopenia, convulsions, hypertension, hypotension, anorexia, constipation, visual disturbances, iodine toxicity (acne, parotitis).

Supplied: Bottles of 50 capsules.

SK&F *Smith Kline & French Laboratories*

Trademark

ORNADE® SPANSULE®

brand of sustained release capsules

Each capsule contains 8 mg. of Teldrin®(brand of chlorpheniramine maleate); 50 mg. of phenylpropanolamine hydrochloride; 2.5 mg. of isopropamide, as the iodide.

UNCOMMON RELIEF FOR COLD SYMPTOMS

OR-203

The girth control pill

Tepanil® Ten-tab®

(continuous release form)

(diethylpropion hydrochloride, N.F.)

When girth gets out of control, TEPANIL can provide sound support for the weight control program you recommend. TEPANIL reduces the appetite—patients enjoy food but eat less. Weight loss is significant—gradual—yet there is a relatively low incidence of CNS stimulation.

Contraindications: Concurrently with MAO inhibitors, in patients hypersensitive to this drug, in emotionally unstable patients susceptible to drug abuse.
Warning: Although generally safer than the amphetamines, use with great caution in patients with severe hypertension or severe cardiovascular disease. Do not use during first trimester of pregnancy unless potential benefits outweigh potential risks.
Adverse Reactions: Rarely severe enough to require discontinuation of therapy, unpleasant symptoms with diethylpropion hydrochloride have been reported to occur in relatively low incidence. As is characteristic of sympathomimetic agents, it may occasionally cause CNS effects such as insomnia, nervousness, dizziness, anxiety, and jitteriness. In contrast, CNS depression has been reported. In a few epileptics an increase in convulsive episodes has been reported. Sympathomimetic cardiovascular effects reported include ones such as tachycardia, precordial pain, arrhythmia, palpitation, and increased blood pressure. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride; this was an isolated experience, which has not been reported by others. Allergic phenomena reported include such conditions as rash, urticaria, ecchymosis, and erythema. Gastrointestinal effects such as diarrhea, constipation, nausea, vomiting, and abdominal discomfort have been reported. Specific reports on the hematopoietic system include two each of bone marrow depression, agranulocytosis, and leukopenia. A variety of miscellaneous adverse reactions have been reported by physicians. These include complaints such as dry mouth, headache, dyspnea, menstrual upset, hair loss, muscle pain, decreased libido, dysuria, and polyuria.
Convenience of two dosage forms: TEPANIL Ten-tab tablets: One 75 mg. tablet daily, swallowed whole, in midmorning (10 a.m.). TEPANIL: One 25 mg. tablet three times daily, one hour before meals. If desired, an additional tablet may be given in midevening to overcome night hunger. Use in children under 12 years of age is not recommended.

I 3325 (2876)

Merrell

MERRELL-NATIONAL LABORATORIES
Division of Richardson-Merrell Inc.
Cincinnati, Ohio 45215

Painful night leg cramps...

unwelcome bedfellow for any patient–including those with arthritis, diabetes or PVD

- ☐ Prevents painful night leg cramps
- ☐ Permits restful sleep
- ☐ Provides simple convenient dosage—usually just one tablet at bedtime

Prescribing Information—Composition: Each white, beveled, compressed tablet contains: Quinine sulfate, 260 mg., Aminophylline, 195 mg. **Indications:** For the prevention and treatment of nocturnal and recumbency leg muscle cramps, including those associated with arthritis, diabetes, varicose veins, thrombophlebitis, arteriosclerosis and static foot deformities. **Contraindications:** Quinamm is contraindicated in pregnancy because of its quinine content. **Precautions/Adverse Reactions:** Aminophylline may produce intestinal cramps in some instances, and quinine may produce symptoms of cinchonism, such as tinnitus, dizziness, and gastrointestinal disturbance. Discontinue use if ringing in the ears, deafness, skin rash, or visual disturbances occur. **Dosage:** One tablet upon retiring. Where necessary, dosage may be increased to one tablet following the evening meal and one tablet upon retiring. **Supplied:** Bottles of 100 and 500 tablets.

Merrell

MERRELL-NATIONAL LABORATORIES
Division of Richardson-Merrell Inc.
Cincinnati, Ohio 45215

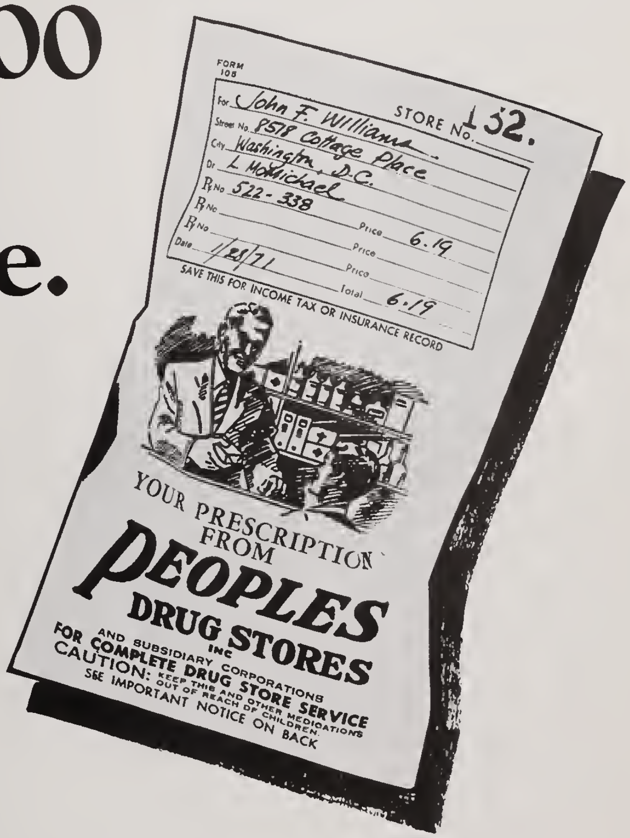
1-3508(3050)

Trademark: Quinamm

Quinamm
(quinine sulfate 260 mg., aminophylline 195 mg.)

Specific therapy for night leg cramps.

The bag that has earned more than 125,000,000 votes of confidence.



FORM 105

For John F. Williams STORE No. 152.
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City Washington, D.C.
Dr L McMichael
℞ No 522-338
℞ No
℞ No
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Price
Date 1/28/71
Total 6.19
SAVE THIS FOR INCOME TAX OR INSURANCE RECORD

YOUR PRESCRIPTION FROM
PEOPLES DRUG STORES INC
AND SUBSIDIARY CORPORATIONS
FOR COMPLETE DRUG STORE SERVICE
CAUTION: KEEP THIS AND OTHER MEDICATIONS OUT OF REACH OF CHILDREN.
SEE IMPORTANT NOTICE ON BACK

Over the years, that's a lot of confidence in prescriptions from Peoples. And we're justifiably proud.

But since we opened shop in 1905, many things have changed. Drug industry research has developed better ways to preserve health and serve the physician. The prescribing physician has developed more sophisticated skills. But one thing has never changed—the careful, professional way in which our pharmacists personally fill your prescriptions.

Filling *your next one* is as important to us as the 125,000,000 that came before it.

THESE 6 PEOPLES DRUG STORES ARE OPEN 24 HOURS A DAY FOR THE CONVENIENCE OF YOU AND YOUR PATIENTS.

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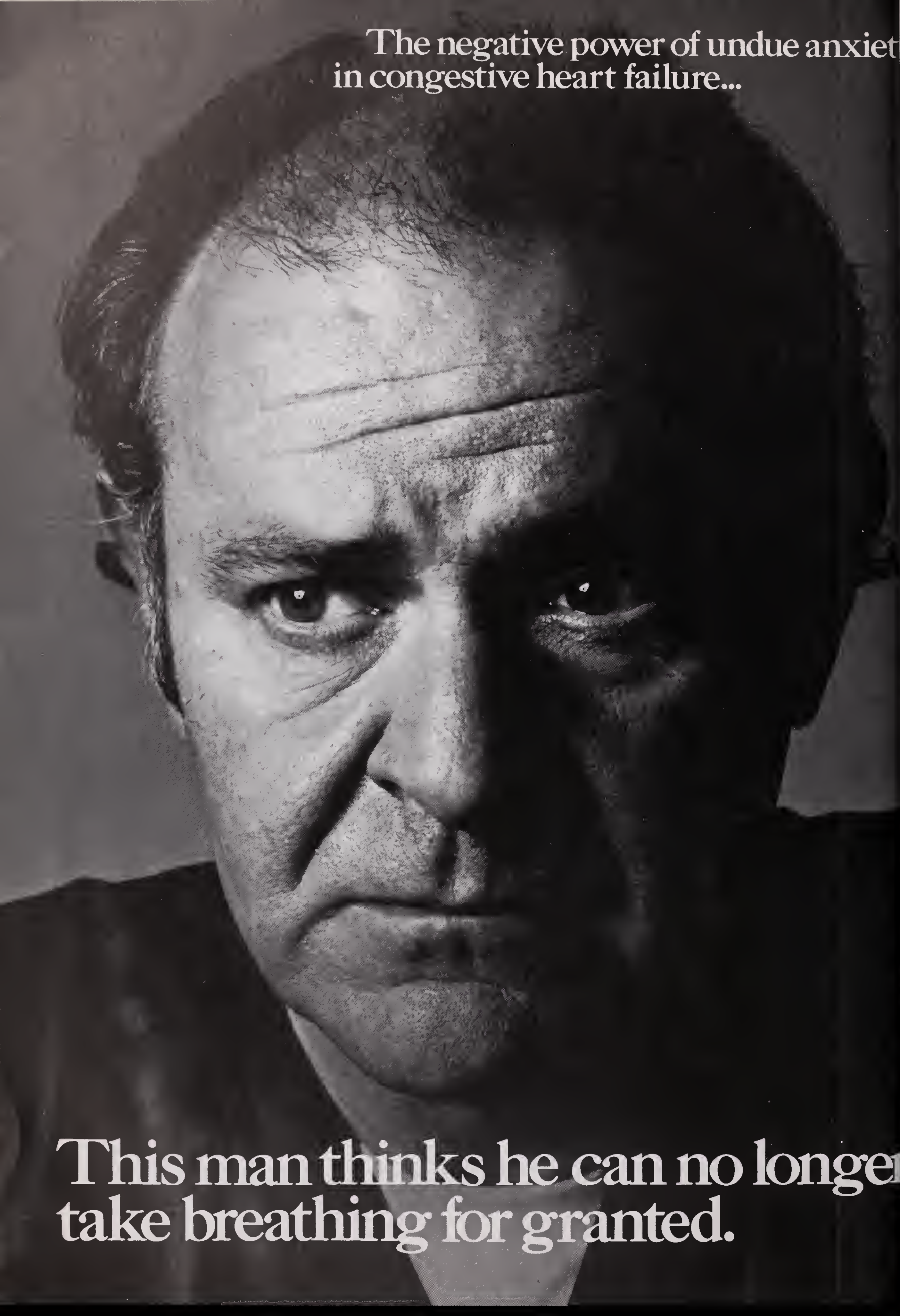
WHEN NEEDED, RECOMMEND A PEOPLES "SAFETY CAP" PRESCRIPTION CONTAINER FOR FAMILIES WITH SMALL CHILDREN.


The negative power of undue anxiet
in congestive heart failure...
This man thinks he can no longer
take breathing for granted.

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Typical of many patients with congestive heart failure, he also suffers from severe anxiety, a psychic factor that may influence the character and degree of his symptoms, such as dyspnea. His apprehension may also deprive him of the emotional calm so important in maintenance therapy.

Aid in rehabilitation
Specific medical and environmental measures are often enhanced by the antianxiety action of adjunctive Libritabs (chlordiazepoxide). Libritabs can also facilitate treatment of the tense convalescent patient until antianxiety therapy is no longer required. Whereas in geriatrics the *usual daily dosage* is 5 mg two to four times daily, the *initial dosage* in elderly and debilitated patients should be limited to 10 mg or less per day, adjusting as needed and tolerated.

Concomitant use with primary agents
Libritabs is used concomitantly with certain specific medications of other classes of drugs, such as cardiac glycosides, diuretics, antihypertensives, vasodilators and oral anticoagulants, whenever excessive anxiety or emotional tension adversely affects the clinical condition or response to therapy. Although clinical studies have not established a cause and effect relationship, physicians should be aware that variable effects on blood coagulation have been reported very rarely in patients receiving oral anticoagulants and chlordiazepoxide HCl.

The positive power of
Libritabs®
(chlordiazepoxide)
5-mg, 10-mg, 25-mg tablets
t.i.d./q.i.d.
up to 100 mg daily
for severe anxiety accompanying congestive heart failure

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Indicated when anxiety, tension and apprehension are significant components of the clinical profile.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported. Use of any drug in pregnancy, lactation, or in women of childbearing age requires that its potential benefits be weighed against its possible hazards.

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Supplied: Tablets containing 5 mg, 10 mg or 25 mg chlordiazepoxide.

ROCHE
Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, N J. 07110

Wellcome

Burroughs Wellcome Co.
Research Triangle Park
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Empirin Compound with Codeine
relieves aches,
pains, fever and
general malaise
of colds and of
FLU

A gratifying announcement about Empirin® Compound with Codeine

CIII

You may now specify up to five refills within six months when you prescribe Empirin Compound with Codeine (unless restricted by state law).

It is significant in this era of increased regulation, that Empirin Compound with Codeine has been placed in a less restrictive category. You may now wish to consider Empirin with Codeine even more frequently for its predictable analgesia in acute or protracted pain of moderate to severe intensity.

Empirin Compound with Codeine No. 3 contains codeine phosphate* (32.4 mg.) gr. ½. No. 4 contains codeine phosphate* (64.8 mg.) gr. 1. *(Warning—may be habit-forming.) Each tablet also contains: aspirin gr. 3½, phenacetin gr. 2½, caffeine gr. ½.

Something new in ampicillin therapy:

low cost

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POTASSIUM HETACILLIN
the ampicillin derivative

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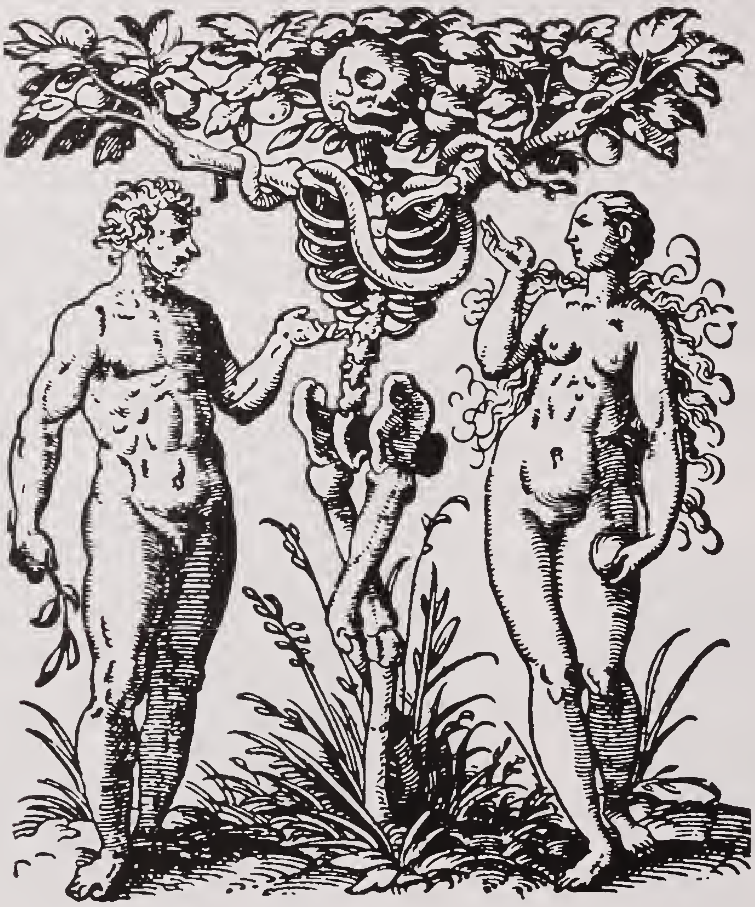
BRISTOL LABORATORIES
Division of Bristol-Myers Company
Syracuse, New York 13201

In acute gonorrhea

(urethritis, cervicitis, proctitis when due to susceptible strains of N. gonorrhoeae)



Sterile Trobicin®
(spectinomycin dihydrochloride pentahydrate)—For Intramuscular injections, 2 gm vials containing 5 ml when reconstituted with diluent. 4 gm vials containing 10 ml when reconstituted with diluent.
An aminocyclitol antibiotic active *in vitro* against most strains of *Neisseria gonorrhoeae* (MIC 7.5 to 20 mcg/ml). Definitive *in vitro* studies have shown no cross resistance of *N. gonorrhoeae* between Trobicin and penicillin.

Indications: Acute gonorrheal urethritis and proctitis in the male and acute gonorrheal cervicitis and proctitis in the female when due to susceptible strains of *N. gonorrhoeae*.

Contraindications: Contraindicated in patients previously found hypersensitive to Trobicin. Not indicated for the treatment of syphilis.

©1972 The Upjohn Company

Warnings: Antibiotics used to treat gonorrhea may mask delay the symptoms of incubating syphilis. Patients should b carefully examined and monthly serological follow-up for least 3 months should be instituted if the diagnosis of syphilis suspected.

Safety for use in infants, children and pregnant women has n been established.

Precautions: The usual precautions should be observed wi atopic individuals. Clinical effectiveness should be monitored t detect evidence of development of resistance of *N. gonorrhoea*

Adverse reactions: The following reactions were observe during the single-dose clinical trials: soreness at the injection sit urticaria, dizziness, nausea, chills, fever and insomnia.
During multiple-dose subchronic tolerance studies in norm human volunteers, the following were noted: a decrease in hemc

Trobicin®

sterile spectinomycin dihydrochloride pentahydrate, Upjohn

single-dose intramuscular treatment

---

ligh cure rate:* 96% of 571 males, 95% of 294 females
)osages, sites of infection, and criteria for diagnosis and cure are defined below.)**

\ssurance of a single-dose, physician-controlled treatment schedule

Jo allergic reactions occurred in patients with an alleged history of penicillin sensitivity vhen treated with Trobicin, although penicillin antibody studies were not performed

\ctive against most strains of Neisseria gonorrhoeae in vitro (M.I.C. 7.5-20 mcg/ml)

\ single two-gram injection produces peak serum concentrations averaging about 00 mcg/ml in one hour (average serum concentrations of 15 mcg/ml present 8 hours after dosing)

Jote: Antibiotics used in high doses for short periods of time to treat gonorrhea may mask or delay the
ymptoms of incubating syphilis. Since the treatment of syphilis demands prolonged therapy with any
ffective antibiotic, and since Trobicin is not indicated in the treatment of syphilis, patients being treated for
jonorrhea should be closely observed clinically. Monthly serological follow-up for at least 3 months should
e instituted if the diagnosis of syphilis is suspected. Trobicin is contraindicated in patients previously found
ypersensitive to it.

)ata compiled from reports of 14 investigators. **Diagnosis was confirmed by cultural identitication of *N. gonorrhoeae* on Thayer-
/artin media in all patients. Criteria for cure: negative culture after at least 2 days post-treatment in males and at least 7 days post-
reatment in females. Any positive culture obtained post-treatment was considered evidence of treatment failure even though the
ollow-up period might have been less than the periods cited above under "criteria for cure" except when the investigator determined
at reinfection through additional sexual contacts was likely. Such cases were judged to be reinfections rather than relapses or
ilures. These cases were regarded as non-evaluatable and were not included.

JA72 1848-6

lobin, hematocrit and creatinine clearance; elevation of alka-
ne phosphatase, BUN and SGPT. In single and multiple-dose
udies in normal volunteers, a reduction in urine output was
ted. Extensive renal function studies demonstroted no con-
stent changes indicative of renal toxicity.

osage and administration: Keep at 25°C and use within
hours after reconstitution with diluent.
lale—single 2 gram dose (5 ml) intramuscularly. Patients with
norrheal proctitis ond patients being re-treated ofter failure
f previous antibiotic therapy should receive 4 grams (10 ml). In
ographic oreos where antibiotic resistance is known to be pre-
lent, initiol treatment with 4 grams (10 ml) intramuscularly is
eferred.
male—single 4 grom dose (10 ml) intramuscularly.

ow supplied: *Vials, 2 and 4 grams—with ampoule of Bacteriostatic Water for Injection with Benzyl Alcohol 0.9% w/v.* Reconstitution yields 5 and 10 ml respectively with a concentration of spectinomycin dihydrochloride pentahydrate equivalent to 400 mg spectinomycin per ml. For intramusculor use only.
Susceptibility Powder—for testing *in vitro* susceptibility of *N. gonorrhoeae.*

Human pharmacology: Ropidly absorbed after intramuscular injection. A two-gram injection produces peok serum concentrations averaging about 100 mcg/ml at one hour with 15 mcg/ml ot 8 hours. A four-gram injection produces peak serum concentrations averaging 160 mcg/ml at two hours with 31 mcg/ml at 8 hours.

For additional product information, see your Upjohn representative or consult the package insert.

MED-B-1-S (LWB)

Upjohn The Upjohn Company, Kalamazoo, Michigan 49001

Arthur los

A single-dose, non-staining anthelmin

his pinworms...

with just one non-staining dose of Antiminth (pyrantel pamoate) Oral Suspension.

Highly effective. Active against pinworm...and roundworm.

Non-staining. Doesn't stain teeth or oral mucosa on ingestion. Doesn't stain stools, clothing or linen.

Simple dosage. Single-dose regimen: 1 cc. per 10 lbs. of body weight (1 tsp. per 50 lbs.).

Well-tolerated. Based on pre-introductory studies.

Pleasant-tasting. Easy-to-take, caramel-flavored oral suspension.

Economical. One prescription for the entire family.

Antiminth (pyrantel pamoate) Oral Suspension

Actions. Antiminth (pyrantel pamoate) has demonstrated anthelmintic activity against *Enterobius vermicularis* (pinworm) and *Ascaris lumbricoides* (roundworm). The anthelmintic action is probably due to the neuromuscular blocking property of the drug.

Antiminth is partially absorbed after an oral dose. Plasma levels of unchanged drug are low. Peak levels (0.05-0.13 μg/ml) are reached in 1-3 hours. Quantities greater than 50% of administered drug are excreted in feces as the unchanged form, whereas only 7% or less of the dose is found in urine as the unchanged form of the drug and its metabolites.

Indications. For the treatment of ascariasis (roundworm infection) and enterobiasis (pinworm infection).

Warnings. *Usage in Pregnancy:* Reproduction studies have been performed in animals and there was no evidence of propensity for harm to the fetus. The relevance to the human is not known.

There is no experience in pregnant women who have received this drug.

Precautions. Minor transient elevations of SGOT have occurred in a small percentage of patients. Therefore, this drug should be used with caution in patients with pre-existing liver dysfunction.

Adverse Reactions. The most frequently encountered adverse reactions are related to the gastrointestinal system.

Gastrointestinal and hepatic reactions: anorexia, nausea, vomiting, gastralgia, abdominal cramps, diarrhea and tenesmus, transient elevation of SGOT.

CNS reactions: headache, dizziness, drowsiness, and insomnia.

Skin reactions: rashes.

Dosage and Administration. *Children and Adults:* Antiminth Oral Suspension (50 mg. of pyrantel base/ml.) should be administered in a single dose of 11 mg. of pyrantel base per kg. of body weight (or 5 mg./lb.); maximum total dose 1 gram. This corresponds to a simplified dosage regimen of 1 cc. of Antiminth per 10 lbs. of body weight. (One teaspoonful = 5 cc.)

Antiminth (pyrantel pamoate) Oral Suspension may be administered without regard to ingestion of food or time of day; and purging is not necessary prior to, during, or after therapy. It may be taken with milk or fruit juices. Because of limited data on repeated doses, no recommendations can be made.

How Supplied. Antiminth is available as a pleasant tasting caramel-flavored suspension which contains the equivalent of 50 mg. pyrantel base per ml., supplied in 60 cc. bottles.

gent: new ANTIMINTH® (pyrantel pamoate)

equivalent to 50 mg pyrantel/ml

ORAL SUSPENSION

ROERIG Pfizer
A division of Pfizer Pharmaceuticals
New York, New York 10017

Among the best sources of iron

ENFAMIL
WITH IRON
READY-TO-USE
INFANT FORMULA
FOR EVERYDAY CONVENIENCE
...JUST POUR AND FEED
32 FLUID OUNCES

12 mg. iron per quart

Routine feeding of ENFAMIL with Iron can help prevent iron deficiency—a serious public health problem among infants of mothers handicapped by their socioeconomic situation. And, ENFAMIL with Iron supplies hungry babies with all the other vitamins and minerals so necessary for proper growth and development. There's no extra cost for the iron. Just extra protection for the infant during the critical first year of life.

ENFAMIL® with iron

Infant Formula provides 12 mg. ferrous iron per quart—at no extra cost

ENFAMIL with Iron Concentrated Liquid in 13-fl. oz. cans,
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ENFAMIL with Iron Ready-To-Use in 32-fl. oz. cans, or
ENFAMIL with Iron Powder in 16-oz. (1-lb.) cans.

Mead Johnson
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PHYSICIANS WANTED–IMMEDIATE NEED. For permanent staff and on sessional basis at PRETERM, 1726 Eye Street, N.W., Washington, D.C. 20006. An innovative outpatient clinic for pregnancy termination, counseling and birth control. Surgical and Ob/Gyn. Call or write Benjamin N. Branch, M.D., Medical Director (202) 298-7300.

AMERICAN UNIVERSITY: Part time physician for Student Infirmary, starting immediately. Afternoon hours. Call 686-2595.

PHYSICIAN NEEDED–full time–A specialty blood donor center supplying pharmaceutical firms with blood products requires the services of M.D. or eligible licensed physician. For specific information regarding responsibilities, compensation, and other pertinent data, please contact Mr. Reilly at Antibodies, Inc., 1712 Eye Street, N.W., Washington, D.C. 298-9084.

PHYSICIAN WANTED: Internist or G.P. 20 hours a week between 9:00 a.m. and 5:00 p.m. weekdays for assessment and screening of adults and consultation to Public Health Field Staff. Salary: $11.33 to $16.37 per hour, depending on qualifications. Call Division of Institutional Care, Prince George's County Health Department, 773-0900.

PRACTICE FOR SALE

FOR SALE: Attractive General Medical practice in McLean, Virginia. X-ray and Pathology Lab in the building. Active office. Desirable lease. Leaving for academic affiliation. Should be able to continue present excellent arrangements for "on call". Box 30, Medical Annals of D.C., 2007 Eye Street, N.W., Washington, D.C., 20006.

GENERAL PRACTICE, established 40 years in Mt. Pleasant area, fully equipped office, X-ray, EKG, diathermy. Call Mrs. E. H. Markwood, (703) 536-7727 or write 4142 N. River Street, Arlington, Virginia 22207.

OFFICE SPACE WANTED

WANTED: Office space for pediatrician. About 225 square feet in area, near Doctors Hospital. Box 35, Medical Annals of D.C., 2007 Eye Street, N.W., Washington, D.C. 20006

OFFICE SPACE AVAILABLE

FOR RENT: 1726 Eye Street, N.W. Ideal office space for G.P. or specialist. Suite for dentist also available. All utilities included plus switchboard and char service. Parking available. Telephone (202) 298-6936.

TO SHARE: Office space Silver Spring busy section University Blvd. West. Convenient to hospitals. Fully equipped internist's office with 2 consultation rooms, 6 treatment rooms, one treadmill testing room. Chem. lab, X-ray, EKG, phone lines, secretary, lab tech. on duty. Happy to show, discuss any timing arrangements, etc. agreeable to both. Might be good second location for one or two interested doctors. Call Dr. Datlow, (301) 593-6010.

BETHESDA MEDICAL BUILDING: Office for lease available immediately, 1068 square feet of well-planned office space in a most desirable location. Free parking for Doctor and staff. Contact Mr. Rosenberg, (301) 652-0983, 8218 Wisconsin Avenue, Bethesda, Maryland.

FOR RENT: 2,000 square feet in Medical Arts Bldg., 3611 Branch Avenue, Hillcrest Heights, Maryland. Modern, air-conditioned, elevator, parking. 4th floor. Rent includes utilities and janitorial service. Call Mr. Cardarelli, (301) 735-7892.

EQUIPMENT FOR SALE

FOR SALE: Laboratory equipment including Bausch and Lomb binocular clinical microscope, Pelton-Crane autoclave and table, Leitz Photrometer. All excellent condition. Telephone (202) 296-8170

OFFICE EQUIPMENT AND FURNITURE, including floor goose neck lamps, American Sterilizer Autoclave, Sklar Portable Suction, soap and towel dispensers, instrument boats, large and small dry sponge jars, Sphygmomanometer (Tycos Taylor), Stethoscope (Littman), ear piercing instrument, Opti Visor Binocular Loop (for use with glasses). Two examining tables, one second century electric and one older model, waiting room furniture including chairs, bench seat with cushion and magazine bench. Please call Mrs. Thomas G. Edison, 949-3434.

FOR SALE: ENT cabinets with suction-pressure units; Beltone Audiometer; examination table; misc. ENT instruments and office equipment—IBM typewriter, Thermofax, Cole modular steel desks and card files. Telephone (703) 533-0360 (Falls Church, Virginia).

FOR SALE: Complete medical office equipment, very reasonable. Includes Haines mechanical sigmoidoscopic examination table, complete surgical instruments, cautery, hyfrecator, vaginal specula, cabinets, office desk, chairs. Telephone DEcatur 2-4848.

FOR SALE: Office equipment including 1 16-inch sterilizer, 2 examining tables, 2 Mayo stands, 2 treatment cabinets, 2 goose-neck floor lamps, 1 stool and 1 chair. Please call: 541-4243 9:00 a.m. to 5:00 p.m. except Wednesday and Saturday.

EXAMINING ROOM AND OFFICE EQUIPMENT, including Hamilton examining table, white with black top; 7-drawer white cabinet; 2 stools, 1 chair, 1 large trash can, all white metal; metal 2-step stool for exercise tolerance test, 1-step metal stool; 1 Burton lamp, 1 Burdick Photomotograph and adapter; a Burdick diathermy model MF49, Profexray fluroscopy; 1 pair lead gloves; Bausch-Lomb monocular microscope and case; other small items. NCR modified one-sixty bookkeeping machine and stand, NCR card tray and cabinet with lock; other card files. Table model Magnavox AM-FM Solid State radio; assorted antique mortar and pestles, "doctor" figurines. Call 223-5000.

FOR SALE: Burdick Model EK-2 ECG, $50; 2 Burdick Kinenometers, $50 each; Picker superficial X-ray therapy, rail mounted. A-O Binocular Microscope with case, $350. 2 examining tables, extremely unusual design, 60 inches long without extension. 1 examining table bed height, hand made, upholstered in black vinyl, best offer; 2 professional ultraviolet lamps (1 Burdick, 1 Hanover); 1 non-motorized dental chair, lead plates for shielding. Contact M. M. Robinson, M.D., 1712 Eye St., N.W., Washington, D.C. 20006 or call (202) 298-7191 or (301) 654-7787 (evenings).

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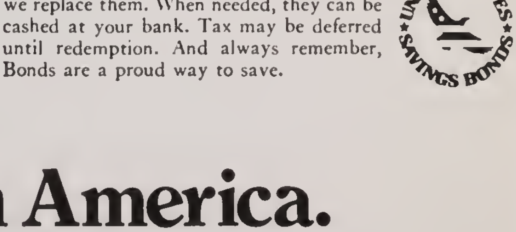

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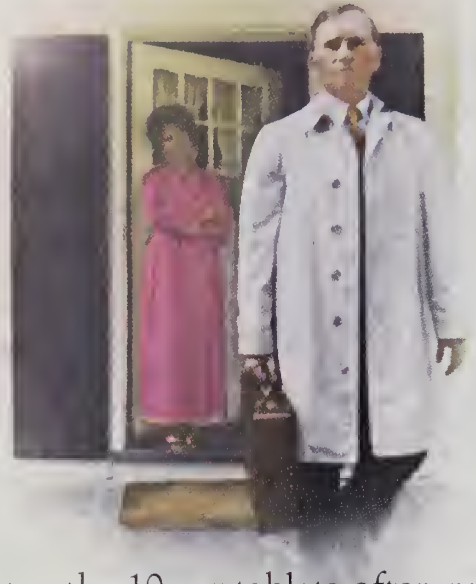

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Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

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> *"I have heard a doctor say on occasion, 'I'm only interested in my profession; I'm not interested in politics.' Let me tell you something: he better get interested in politics or he won't have any profession to be interested in."*
>
> Richard Nixon

Darrell C. Crain, M.D.

A blunter warning or one from a more authoritative source could scarcely be found. The message is clear and rather disturbing: elect legislators who will listen to the voice of medicine this year or see the practice of medicine as we now know it, doomed. And there is only one way to elect such legislators - to contribute time and money.

This is the place for forthright statements, not pious platitudes or circuitous generalizations. During the past year our Society became involved in politics, of a purely parochial nature. But it brought results and served as a model for what we must do during the coming year on a broader scale. Specifically, our action was twofold. The Society employed a legislative assistant (the euphuistic term for "lobbyist"). He shepherded the Bill on Incorporation through Congress, getting a member to introduce it, rounding up legislators when voting time came and generally presenting the Society in a favorable light. He is currently working in a similar manner on other bills our legislative committee has endorsed: licensure, medical records, blood and tissue, and malpractice.

The other phase of our action has been and will be even more important, namely, campaign contributions. All the Congressmen and many of the Senators who supported our cause are up for reelection. Running a campaign is an expensive affair. They have a right to anticipate our support. And we must broaden our horizon to support legislators who think rationally rather than emotionally or politically on the entire spectrum of legislation as it relates to government planning, financing, monitoring and controlling health services.

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It is not enough to say that we will vote for men in November whose announced policies are acceptable to us. We must contribute now to their campaigns. It is of course difficult for each physician to study the complexities of the numerous bills and to make meaningful financial contributions to all legislators deserving of support. But in politics, as elsewhere, in union there is strength. So William Felts, Chairman of our Committee on Legislation, is working with Karl Jonas, Chairman of DOCPAC in determining just where our dollars can best be invested. This cannot be Society money - rather it comes from individual subscribers. And many of our members have not yet joined the only group dedicated solely to the election of men sympathetic to our cause. The minimum membership contribution is $20. If you feel you might just forget the whole thing and save $20, it might just be the last money you ever save! But it could turn out that your $20 investment brought the greatest percentage gain of your life!

Do it now - write Karl Jonas - join DOCPAC.

Darrell C. Crain

GEORGE WASHINGTON UNIVERSITY
MEDICAL STUDENT RESEARCH NUMBER

This issue of the Medical Annals of the District of Columbia underscores the changing relationship between the medical student and his diplomaed colleagues; that is, the acknowledgement of the student as an active participant in today's educational schema. Implicit in this issue is the recognition of the continuity of student to physician and the corollary - that good physicians are life-time students.

In Doctor Harold Stevens' article, "Student Research - Preparation for Practice," he emphasizes the enduring historical contributions that have been made by many physicians, while still neophytes in their career. It is this ingenuity, fresh approach and new look at old subjects that characterizes the first issue of the Medical Annals of the District of Columbia devoted exclusively to student research.

Who among us can not learn from our student colleagues?

Raymond Scalettar, M.D., F.A.C.P.

WOMEN IN MEDICINE

The United States falls far short of other countries in educating women for medicine. Women physicians comprise only 7.6 per cent of the physician population of this country, and that only because more than one-third are graduates of foreign medical schools. But indications are that traditional prejudices against women physicians in this country are gradually eroding.

According to a recent study, the total physician population in this country as of December 31, 1970, was 334,028 including 25,401 women (1). Contrary to widely held opinions that women do not use their professional education, the same report showed that 83.9 per cent of all women physicians were active in medicine at the time the data were compiled. Nearly one-third of the inactive women, presumably those in the younger age groups, indicated that they were only temporarily not in practice. In other words, more than 88 per cent of all women physicians in this country were or intended to be in medical practice of one form or another. Most of the inactive women were retired because of age or disability. Only 4 per cent were inactive for unexplained reasons.

A survey by the Association of American Medical Colleges showed that in the school year 1971-1972, when a record enrollment of 12,361 first-year students occurred in the 108 U.S. medical schools, 13.5 per cent or 1,673 were women. For the year 1968-1969, only 887 or 9 per cent of the first-year classes were women (2).

The American Medical Association, long indifferent to women in medicine, has also taken a more encouraging attitude. In June 1971, the AMA House of Delegates adopted a report in which these statements appeared:

> "AMA believes there is a need for more women physicians...Women seeking a career in medicine face discouragement and handicaps...Changes are needed to increase the number of women physicians ...Specific changes necessary are: (1) increased financial assistance for men and women, (2) continuing emphasis on flexible scheduling during medical school years, (3) flexible scheduling during the internship and residency years, and especially (4) a social environment more conducive toward the fuller utilization of talented women in many careers. Many more young girls will need to be given a different set of goals for their adult lives. They must be encouraged to prepare themselves for medicine and to apply to medical schools...(3)"

While discrimination against women in medicine is lessening, equality is far short of realization. How much better it would be to arrive at the necessary changes through conviction that women are needed in medicine rather than by legal actions under equal opportunity laws that women activists now contemplate. Time is running out for medical schools that still limit their enrollment of women to 5 or 6 per cent.

Happily, the three medical schools in D.C. have been among the leaders in increasing the enrollment of women students. Moreover, nearly 12 per cent of all physicians in the District are women, well above the national figure of 7.6 per cent.

Josephine E. Renshaw, M.D.
Past President
American Medical
Women's Association

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3. Board of Trustees Report O: Physician Manpower and Medical Education. Proceedings House of Delegates, June 20-24, 1971. Chicago: American Medical Association, 1971, 78.

CORONARY BYPASS SURGERY

At last! or Again?

> *"It's not so much what we don't know that causes trouble as what we know, that ain't so."*
>
> Will Rogers

Coronary heart disease, the greatest incapacitator and killer of adult Americans, has no cure. Medical therapy offers much in terms of control of complications and symptoms but has not made any impact on preventing or reversing the fundamental atherogenetic process. The stage is set for the acceptance of any new approach, but especially one that offers the appeal of the quick, the simple and the logical.

Is saphenous vein bypass of stenotic proximal coronary arteries the answer we have been patiently waiting for? Certainly the concept of bypass surgery to revascularize the lower extremities has proven valid. The surgeon's ability to anastomose a vein graft to a tiny artery in the heart has been demonstrated. Significant blood flow in such grafts have been recorded on the operating table, presumably providing immediate revascularization of the ischemic portion of the heart. The mortality rate of such surgery has been quite low, at least in the large centers reporting, and accounts of marked improvement of angina in most patients, glowing.

Is it possible that bypass surgery is too quick, oversimplified and illogical? We cardiologists must be understood for reacting with instinctive caution, if not skepticism, to any new surgical approach. This attitude has been conditioned by 30 years of revascularization procedures that have exploded onto the scene supported by anecdotal and testimonial "evidence" of success. The epicardial poudrage, internal mammary artery ligation, endarterectomy, patch graft and internal mammary implantation procedures all were supported by claims of improvement in angina in 80-95 per cent of patients. All have left or are fading from the scene because of unacceptable mortality or proven lack of benefit. Only one of them, the internal mammary ligation, was subjected to a controlled trial (only after it was being freely performed as a clinically acceptable treatment) and fortunately dispensed with quickly. Now we are again faced with a therapeutic technique that is sweeping the country without a controlled evaluation of its benefit. It is currently unthinkable and impermissible to introduce a drug to the public without rigidly designed, strictly controlled trials and statistically convincing results, demanded by the FDA, granting agencies and responsible journals. The demonstration of technical feasibility, the simplicity of the mechanical approach and even the improvement in flow dynamics, are not to be equated with net benefit to the patient. Already there are reports of occlusion of 20-40 per cent of the grafts within two years. It may be argued that this only leaves the patient back where he started from, having gained a period of symptomatic benefit. However, there is one study showing the post shunt development of complete occlusion of the previously only stenotic proximal coronary artery segment. If this should prove the rule, then the patient whose graft becomes occluded is worse off than before. On the other hand, if bypass surgery is logical and long lasting it should prevent myocardial infarction and prolong life. How can we ever know the answers, particularly in as variable a disease process as coronary heart disease, without a controlled trial? The Veterans Administration Cooperative Study is the only one in which each operated patient has a non-operative control of the same age group and same anatomic involvement. Past experience is certainly not adequate control. Prospective natural history information is needed in patients whose exact anatomic extent of coronary disease is known by coronary arteriography. Perhaps the best surgical candidate is also the patient who needs it the least. (For example, the Johns Hopkins study by Friesinger et al (1) indicates that the young man with a single discrete proximal stenosis in the anterior descending has an excellent prognosis, on medical management)

Where does this leave the practicing physician, buffeted by controversy and pressured by headlines? I believe that he needs to tread a course between the extremes of withholding surgery from everybody, pending a definitive answer, and recommending surgery for all patients with anatomically "suitable" lesions. It might be reasonable to consider a patient a candidate who is significantly incapacitated by angina, despite medical management, to the extent that he finds the necessary alteration of his usual life style unacceptable; and whose coronary arteriogram shows a discrete, proximal, severely stenotic lesion in one or more major coronary arteries with angiographic visualization of a good distal vessel; and whose ventriculogram shows normal ventricular function. Furthermore, he should obviously refer his patients to an institution totally prepared with appropriate catheterization laboratory, surgical facilities, cardiologic, surgical, nursing and paramedical personnel, and where cardiovascular surgery is performed on a regular basis. Such institutions should insist on careful scientific, objective followup studies such as metabolic, hemodynamic and angiographic, as well as clinical evaluation.

It may well turn out that coronary bypass revascularization will be beneficial to at least a well defined limited segment of the coronary disease population.

It would be tragic if this were to be obscured by premature overenthusiastic indulgence in surgery on poorly selected patients or in poorly prepared hospitals.

> *"From principles is derived probability, but the truth or certainty is obtained only from facts."*
>
> Nathaniel Hawthorne

Gerald I. Shugoll, M.D.
Chief, Cardiology Section
Veterans Administration Hospital

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BY-STANDING INNOCENTS

The Children's Crusade Revisited

The opinions and actions of a great many smokers notwithstanding, the nonsmokers are and always have been very much in the majority. Of the 203,000,000 people in this country 65,000,000 are less than 18 years old. Less than one-third of the population smokes (28 per cent, in fact, using the figures that 51 per cent of men over 17 are smokers and 33 per cent are women). The nonsmoking men can presumably fight their own battles, and a share of the women who do not smoke are militants - but few indeed have championed the cause of the children.

This is not true of the unborn child, who has not been quite so ignored. Low birth weight and increased neonatal mortality in the infants of smoking mothers drew attention years ago, and although there is still argument, the 1971 Surgeon General's Report on "The Health Consequences of Smoking" states that "There is strong evidence to support the view that smoking mothers have a significantly greater number of unsuccessful pregnancies due to stillbirth and neonatal death as compared to nonsmoking mothers. There is insufficient evidence to support a comparable statement for abortions. The recently published Second Report of the British Perinatal Mortality Survey, a carefully designed and controlled prospective study involving large numbers of patients, adds further support to these conclusions (1)." While the Surgeon General's Report cites some 36 references from the literature in regard to pregnancy and the fetus, it does not even index the effects of smoking on children who do not smoke, and MEDLARS (computerized literature search produces only two titles (2,3); the Surgeon General's Report a third, in a group of epidemiologic studies (4); and cross references a fourth (5).

The belated recognition of the effects on the bystander of so visible and malodorous a pollutant is surprising, but these effects are newsworthy enough at the moment to very recently rate presentation in Time magazine. Nevertheless, most of us have been at least dimly aware that smoking had deleterious effects on children in smokers' families. Cameron, using a direct and ingenious approach, surveyed 1,000 Denver families and in a more polished later study surveyed 727 households in Detroit. The questions concerned the age, sex, and relationship of household members; their illness in the last week (early spring), the nature of the illness, and its severity; who smoked and how much; and whether there was an automatic dishwasher in the home (as a socioeconomic index). The results are impressive even though the authors consider them very suggestive rather than definitive: there are more ill children in homes with smokers, with the preponderance respiratory diseases, and more exposure produces more illness (2,5).

May we add the rights of children to the rights of the smoker?

Robert B.W. Smith, M.D., F.A.C.P.

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THE ETERNAL TRIANGLE

Is a serum cholesterol an "indicated" test procedure in a patient with hypertension? Is an electrocardiogram an "indicated" procedure in the patient with abdominal pain? Is a chest x-ray an "indicated" procedure in a patient with rheumatoid arthritis?

Most physicians would probably answer the above questions in the affirmative; yet some of these judgments are being questioned by insurance carriers because, they say, the contracts are written for "the diagnosis and treatment of disease." Unless the individuals reviewing all claims for insurance are physicians or unusually well-qualified laymen, they can hardly be expected to appreciate the neurologic pathways that sometimes refer cardiac pain to the abdominal region, and can hardly be expected to be aware of rheumatoid pulmonary and pleural disease. Similarly, the physician and his staff at the other end of the insurance claim line are forced to render finer and finer judgments as to when a given procedure represents a diagnostic test for a specific disease and when it represents a routine part of periodic physical examination. The whole process reminds me of experiments in which animals are forced to discriminate between a circle and a series of ovals that more and more approximate circles. The result is a neurotic, immobilized animal.

In order to mitigate the onerous task of making these fine distinctions, most physicians simply avoid the issue and list all diagnoses and all tests performed and leave it to the carrier to decide which tests are reimbursable for those given diagnoses. This too is unsatisfactory, since it leaves the decision-making entirely to the employees of the insurance carrier. They may then provide underpayment or overpayment to the patient. And the patient, the most important corner of our triangle, is least of all in a position to know whether he is being treated fairly or not.

I would like here to propose a partial solution to this problem. Why not provide full or major coverage for a good, complete routine physical examination at regular intervals? The extent and frequency of the examination could vary, depending on the patient's age, sex or other factors. After all, most knowledgeable patients are already getting periodic examinations and, since most patients do have symptoms of various sorts, one would suspect that for the most part, insurance carriers are already paying for them.

It is difficult to suggest changes that smack of broadening coverage in these times of escalating premiums and increasing deficits in many insurance programs. However, I would think that this suggestion might be quite actuarially sound, particularly if a co-insurance factor were included. Furthermore, a great deal of effort has gone into convincing the public that early diagnosis and periodic physical examinations reduce the ultimate cost of health maintenance; such a measure would encourage people to have such examinations.

But whatever else is or is not accomplished, at least this innovation would take some of the nitpicking out of the already strenuous art of claims filing.

Paul Schlein, M.D.

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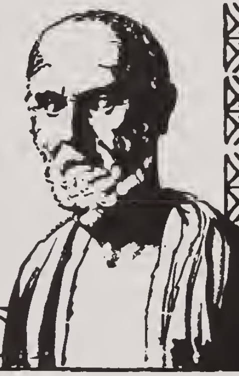

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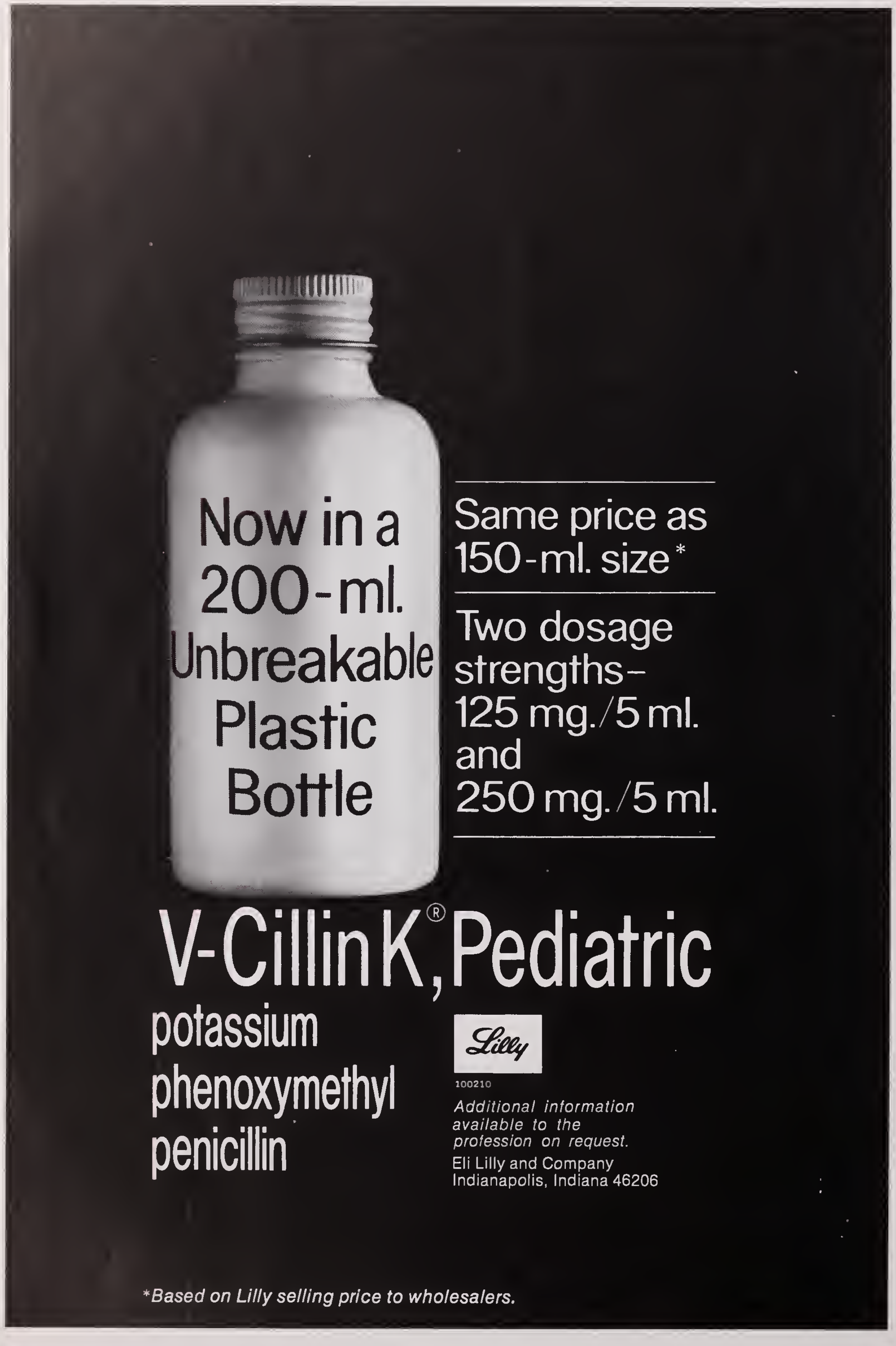

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Student Research—Preparation for Practice

HAROLD STEVENS, PH.D., M.D.*

"Youth will have his course"
John Lyly, 1579

Viewed in perspective, medical education, like other historical evolutions, shows cyclic patterns. The freewheeling, unstructured, polymorphic programs leading to a medical degree at the beginning of this century may be returning, transmogrified and in an altered context, but with many similarities to the pre-Flexnerian era. The Flexner report, which sharply defined the objectives and methodology of medical education, forged the template which then molded all, stamping out a uniform product for 30 years before the current decade of discontent was upon us. The tight uniform casing of the universal curriculum is now folded, spindled, and mutilated. Distraught medical educators, finding their frenzied patchwork unsuited to the turbulent time, were driven to weave a tangled web in which today's student is often more enmeshed than supported.

The resulting open-ended permissive curriculum is a two-edged sword, cutting the student loose from the prior rigid lock-step formation, offering wide freedom of choice, but often failing to provide adequate faculty contact and direction to identify and channel his individualistic needs. Boredom, discouragement, cynicism, and poor preparation for practice may result.

A partial resolution of this doctors' dilemma may be found in the student research programs offered with increasing acceptance in many medical schools. The usual format consists of an annual meeting where students present the results of their research. The ceremony is concluded with awards for superior achievement and an address by an eminent scientist.

But meritorious student research projects demand a large segment of the student's elective time, and according to James' law of conservation of the curriculum–whenever you add something fresh to the curriculum, you must take something out. [1] Is the elective time required by the student to pursue such research worth the time and effort? Or would it be more constructive for him to participate in conventional didactic exercises? No unambiguous answer is now possible, but insight into the value and outcome of student research can be gained by reviewing the past.

The model of meritorious student research most often cited is the contribution of Charles H. Best. The groundwork was laid by another medical student, Langerhans, who discovered the "islets" 2 years before he was graduated from medical school. And it was a laboratory assistant in Minkowski's laboratory who observed flies gathering around the sugar-laden urine of the pancreatized animals. [2] William George MacCallum, another student, performed the crucial experiment linking islet-cell disease with diabetes. The field of metabolism has been enriched by other student investigators, including Frederick Gowland Hopkins who, as a student, published a paper on the pigments in the wings of English brimstone butterflies. He found the pigment to be a derivative of uric acid and in his typical way immediately improved the method for the determination of uric acid. The study of calcium metabolism in teeth and bones led the Swedish medical student, Ivar Sandstrom, to discovery of the parathyroid glands. His paper was returned to him by a German editor for being too long to publish.[3]

Not all student research was or is performed in elegant laboratories with sophisticated equipment. Witness Jenner who in 1768 at age 19, astutely listened to the milk maid's story and drew the inference that eventuated in his classic experiment and launched a lifelong pursuit of smallpox prevention.

John Shaw Billings, an impoverished medical student, forced to live in a city hospital in Cincinnati, studied hospital administration and its defects, thereby establishing the foundation for his subsequent massive contribution to improved patient care. In his first army post in the Civil War, Billings excoriated Cliffborne General Hospital in Washington, D.C. as being "in an extremely filthy and dilapidated condition" (Fig. 1).[3]

Even allowing for my personal bias, there appears to be an inordinate number of student investigators who have been fascinated by neurological problems, among them the talented Billings, who as a student attempted a statistical analysis of the results of the surgical treatment of epilepsy. This investigation required laborious and inefficient search of books in various libraries in several cities over a period

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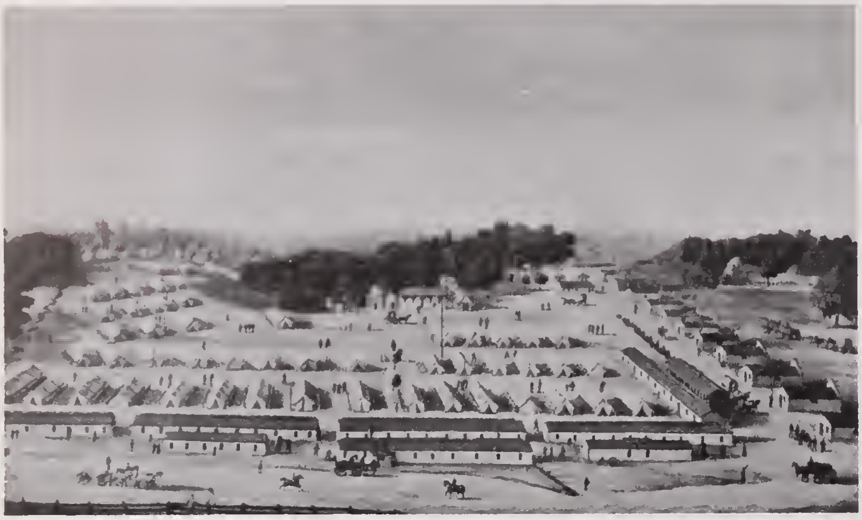


FIG. 1. Cliffburne General Hospital, Washington, D.C., 1861.

of 6 months. This frustrating experience as a student fueled his later heroic and successful efforts that culminated in the foundation of the Index Medicus. Neurology has been a beneficiary of many other student investigations, including those of Pavlov, Cajal, and Sherrington, all of whom in later life were awarded the Nobel prize. Several neurological syndromes were identified by student investigators, including Korsakow's syndrome, Brown-Sequard syndrome, Huntington's chorea, Schilder's disease, and Klumke's paralysis. The last was described by Augusta Klumke, who later married another neurological student investigator, Joseph Dejerine.[4] Peroneal muscular atrophy was identified by Howard Tooth and was the subject of his doctorate thesis (Fig. 2). His name is joined with Pierre Marie, another student investigator in the eponymic designation of the Charcot-Marie-Tooth syndrome.

A review of titles of papers presented in 1970 by students at several medical schools discloses that the thrust of modern student research, unlike that in the past, is generally more basic and at times even esoteric, inviting the criticism that "practical research" has been neglected.[5] Even louder complaints may be voiced now that government student summer support and other research subsidy has been curtailed. There is danger that student research interest and activity may falter or be labelled wasteful, starry-eyed dilettantism.[6]

This old and universal controversy over basic versus applied research recalls of Benjamin Franklin's response when, while as ambassador to the French court, he witnessed some early balloon ascents. In reply to sceptics who queried what use a balloon might ever have, Franklin is said to have replied "What good is a newborn baby"? Also Michael Faraday, who discovered electro-magnetic induction, the foundation of modern electric power, was demonstrating this phenomenon in his laboratory to Prime Minister Gladstone. His distinguished visitor, who was unimpressed, asked, "What is the good of this discovery"? Faraday is said to have replied "Someday, sir, you will collect taxes from it".

Admittedly, this marked preponderance of basic science subjects on the student programs reflects the changing emphasis in clinical research. This observation was recently quantified in a study by Feinstein et al [7,8] and implies a need to broaden future research opportunities for students in order to captivate the interest of a larger segment of the student body which in recent years grows ever more heterogeneous in temperament, in social, ethnic, and intellectual background, and in objectives and motivation.

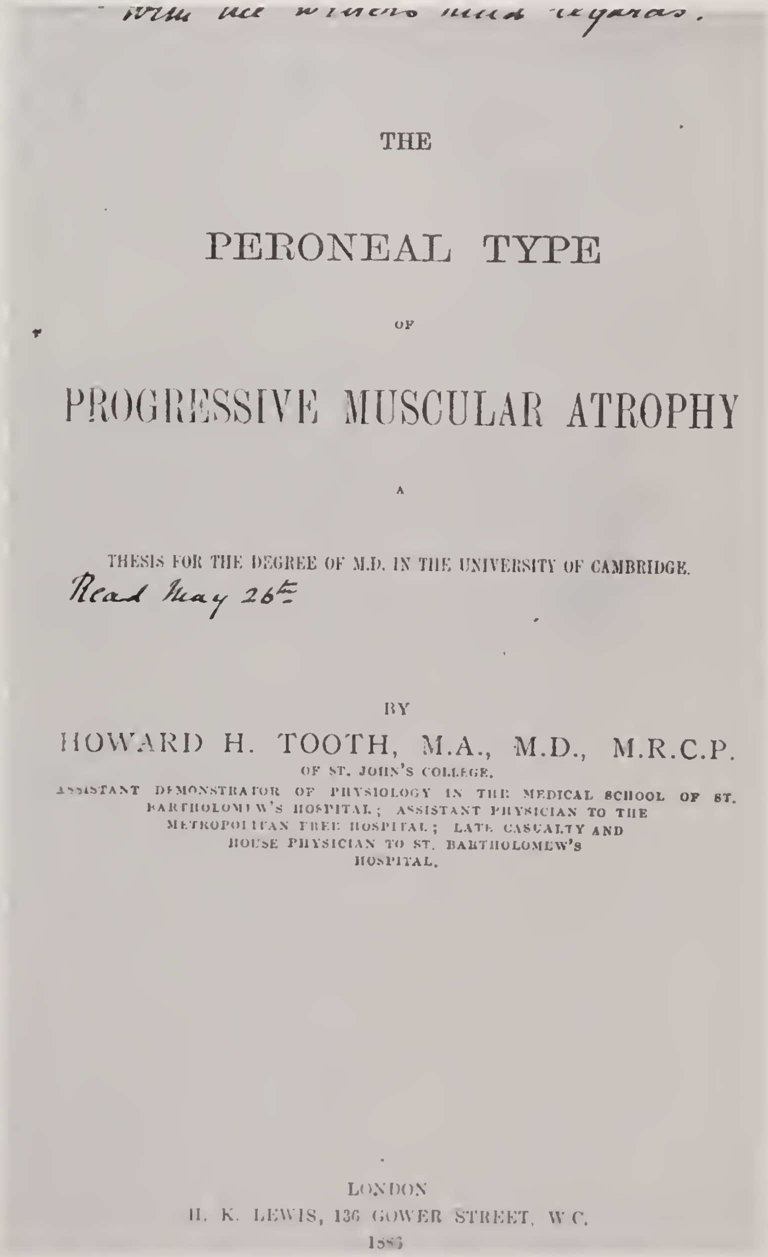
Although collaborative investigation, either in the university, the community hospital, or in the group clinic,[9] is generally acknowledged to be the most effective pattern for research, the talented individual working alone and, perhaps because he is working alone, unfettered, unsupervised, uncommitted, and often without elegant equipment, can make spectacular scientific contributions. The most revolutionary hypothesis in the last century and a half, the evolution of species by natural selection, was born out of a 1-man study of animals and plants in the Galapagos Islands by Charles Darwin, and of animals and plants in the islands of the East Indies by Alfred Russell Wallace [10]. The solo investigator is not extinct.

In 1961, when Houssay received the Nobel prize he stated, "The most remarkable piece of research apparatus is the human brain. Some people want to buy every piece of equipment known to science. They believe that with a beautiful building filled with modern equipment, they have a first-rate research institute. This is a superstition. The great discoveries have been made by men working alone."[11]

No man is an island, but medical educators should appreciate that the individual investigator is not extinct and that the talented individual's curiosity and drive to discover is irrepressible; it is so compelling that, even in this day of complex, elegant laboratories, the gifted solo investigator, even in a field different from his own, may make substantial contributions to science and technology.[12] The automatic dial telephone was devised by a mortician. The inventor of xerography was a lawyer. The pneumatic tire came from a veterinary surgeon. Does specialized training so cramp imagination that young audacious investigators are compelled to apply their inventive genius only in fields where they have not learned the obstacles? The eager but ingenuous student, working alone or with a senior collaborator, may perceive solutions or devise novel attacks on a problem that are unconventional and impractical, born of naivete that is impervious to logic but workable; witness the consequence of Marconi's obdurate and absurd rejection of the established law of physics that radio waves could not project beyond the horizon.

The student can be encouraged to experiment anywhere, not only in the laboratory. A single case study is often a valid research subject. The field need not be biological. Many students are intrigued by problems in the distribution of medical care and other medical-social issues. In any context, students can be offered the chance to exchange ideas freely in intimate, informal communication, to benefit from what the philosopher Josiah Royce labeled the "fecundity of aggregation".

Encouragement of student research has value beyond whatever contributions to science may result. It is currently the most effective tactic for establishing a one-to-one relationship of student to faculty member, a need that grows with enrollment. Furthermore, the brimming knowledge, enthusiasm and unorthodoxy of students who tackle research are powerful forces, serving both to enhance investigative productivity and to catalyze faculty research.



With the writer's kind regards.

THE

PERONEAL TYPE

OF

PROGRESSIVE MUSCULAR ATROPHY

A

THESIS FOR THE DEGREE OF M.D. IN THE UNIVERSITY OF CAMBRIDGE.

Read May 26th

BY

HOWARD H. TOOTH, M.A., M.D., M.R.C.P.

OF ST. JOHN'S COLLEGE.

ASSISTANT DEMONSTRATOR OF PHYSIOLOGY IN THE MEDICAL SCHOOL OF ST.
BARTHOLOMEW'S HOSPITAL; ASSISTANT PHYSICIAN TO THE
METROPOLITAN FREE HOSPITAL; LATE CASUALTY AND
HOUSE PHYSICIAN TO ST. BARTHOLOMEW'S
HOSPITAL.

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FIG. 2. "Peroneal muscular atrophy was identified by Howard Tooth and was the subject of his doctorate thesis."

Aside from offering a broad spectrum of opportunities encompassing the varied interests of the largest possible segment of the student body, there is no stereotyped formula for encouraging research nor for identifying the promising student-investigator, a compelling obligation for the faculty.

How does one recognize the young gifted investigator? This crucial question is further compounded by the recent recognition that there are 2 basic types of research activity: 1) problem-finding, defined as the detection of the need for a new program by comparing existing and expected future programs, and 2) problem-solving, defined as the selection and use of an existing program from an existing set of programs.

The problem finders are rare, and their rarity is a main bottleneck in scientific progress. According to Mackworth[13] 1 reason why problem finders are rare is that aptitude and other psychological tests are biased against problem finders. They inevitably favor the solver. So long as educational psychologists use examinations and intelligence tests which select efficient verbal reasoners rather than the intuitive observers or constructive thinkers, the problem finders will be left to their own devices. Here is another reason why criteria for selecting medical students need systematic re-examination and validation.

The Flexner model of the premedical and medical student is consonant with the quiet dogmas of the past but is no longer adequate for the stormy present. Indeed, history records some unlikely candidates for admission to medical school who subsequently were admitted to the Pantheon of medical history. Cajal was jailed for constructing a cannon from a hollow tree and blowing down the wall of a grandee's palace. This juvenile delinquent became a Nobel prize winner. John Hunter could not read or write at age 17 and was a furniture maker until age 20. Claude Bernard, another student investigator, was an average medical student who barely passed the competitive examination for internship.[14] Paul Ehrlich got through his final medical examinations only by the grace of the examiners who recognized his special talents. Hughlings Jackson was indifferent to schooling in his early years,[15-17] and Walter Cannon was removed from school by his father because he was inattentive.[18]

These and many other talented student investigators were salvaged by the interest, patience, and the inspirational talents of great teachers. For example, Cannon was set on fire by Bowditch, who was inspired by his teacher, Carl Ludwig, one of the most esteemed teachers in history, as was Johanes Mueller who nurtured such giants as Helmholtz, Remak,[19] and Gruby.[3] The last 2 overcame unusual obstacles in their dedicated efforts to study and learn.

Conversely, many able minds have been lost by insensitive or indifferent teachers, e.g., Charles Darwin left Edinburgh Medical School after 2 terms because of crushing boredom. As competition grows keener among disciplines for recruiting the gifted, medical faculties would do well to study the care and feeding of these valued people. Again history warns us that costly leakage has occurred. A large number of talented intellects have been lost by medicine to the field of literature. Hughlings Jackson was nearly one of those. Claude Bernard, Gilles de la Tourette, and Henri Claude were unsuccessful playwrights. Gilles de la Tourette continued to have a parallel career as a writer while he distinguished himself as a neurologist, medical educator, investigator, and health administrator[20,21]; medicine lost Borodin and Berlioz to music; Freud, Oliver Wendell Holmes, and Semmelweis nearly defected to the law; Pierre Marie was actually admitted to the bar; Osler originally planned a career in the ministry, as did Nissl and Donders; Purkinje was actually a noviate for 3 years; Von Economo abandoned engineering. All had been student investigators.

As stated, many were directed to or were held within the orbit of medicine by the attraction of an inspiring teacher. Langerhans had his Virchow who had his Mueller; Jenner had his Hunter, and Gruby, Rokitansky. Pierre Marie was stimulated by Charcot, MacCallum by Osler and Simon Flexner.

The lesson is clear. We need imagination and tolerance in place of the past mindless inflexibility, perhaps better labelled decerebrate rigidity. Above all, we need teachers who inspire the gifted and recognize the hidden talents of the rebellious and unorthodox. Student research programs may be the vehicle or modus operandi for reaching out and establishing contact with the young and for providing the much needed and neglected moral leadership. Close contact with an exemplary teacher-investigator has favorably fashioned the value system of many a student. Nor is this interaction one way. The presence of the malleable student imposes a high standard of ethical response on the senior investigator. The idealism of the young, starry-eyed and evanescent though it may be, is sincere and impressionable. Youth genuinely abhors the materialism of their elders and would agree with Harvey Wheeler[22] and others that money and power have corrupted the sciences and professions, making it appear that the only people capable of maintaining an ethic for a profession are the young before they have used it to become rich and influential. This is consistent with Harold Laski's proposition that the effective regulator of the American Judiciary are the law journals which are run by the young before they have made any money practicing law. Harvey Wheeler suggests that we institutionalize the critical and ethical talents of youth and focus them on the conduct of the sciences as is now done for the judiciary by the law journals. This problem is actually quite ancient. Aristotle formulated it clearly, "Youth loves honor and victory more than money; it really cares next to nothing about money for it has not yet learned what the lack of it means".

The military-industrial-academic complex has turned many students off. They see that the money changers are in the temple of learning and have converted it to a tower of Babel.[23] Too often the modern-day professor has turned entrepeneur; his booty may include a fat salary from a drug firm, eager to wear that professional look, or a juicy fee as a consultant, or a federal grant big enough to support him and a staff of associates. Faced with this massive corruption of what was once the purpose of a professor, the student joins the revolt against the Establishment.[24] He is as outraged as a zealot of the Reformation period faced with the massive corruption of the Church.

The systematic search for sources of funds has subverted at times the original purpose of the university almost to the point where Willie Sutton becomes an emulative figure. On occasion, professors, preoccupied with missions to Mammon, have farmed out fragments of their pet research projects but spent as little time in the laboratory with the student as they did in the classroom. This technique will subvert the student research program and alienate the student who clearly sees the connection between the allocation of Federal funds for a specified problem and sudden spontaneous generation of investigators in that specified field.[25] Admittedly, the problems of financing medical research have reached crisis proportions, succor from any source is welcome, and researchers will go where the money is, not unlike the seduction-prone lady from Kent:

There was a young lady from Kent
Who said that she knew what it meant.
When man took her to dine,
Gave her cocktails and wine,
She knew what it meant, but she went.

The reports published in this issue of the Annals testify to many principles, including the constructive benefits of student-teacher collaboration and the unique research opportunities available in the Washington area. The student authors, if history serves us, have a good prognosis as doctors and citizens if not as scientists. While many of their peers were blindly assaulting the bastions of the Establishment, physically and verbally, or singing protest songs on electric guitars, these gifted and goal-directed students were assaulting the sequestered secrets of nature, prying fragments, however small from the limitless mass of the unknown. Their efforts deserve recognition and reward. This does not mean that student research should be compulsory; it should be made attractive, not as a self-justifying endeavor, but to prepare the student for the practice of medicine. For the capable student-investigator is also capable in the clinic since he intuitively recognizes that every patient is an individual challenge. He approaches each diagnostic puzzle with a sense of intense personal involvement and excitement, using the same techniques of careful observation, and information assembly, weighing and analyzing the evidence. And, in either the laboratory or in the clinic, he finds the joy of engaging in intellectual exercise, and soon discovers that the search for truth is more important than its possession.

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Studies on the Nerve Endings in Human Hearts*

EDISON H. MIYAWAKI, M.S.† AND DONALD H. GLEW, JR., M.D.‡

During the past century neurohistologists have described, named and classified a large variety of nerve endings in the heart of lower animals. Major contributions to the subject of nerve endings on myocardial muscle fibers in lower animals have been made by Gerlach (1866),[1] by Dogiel (1898),[2] and in recent ultrastructural studies by Fawcett[3] and Whittaker[4].

But, for some unknown reason, such prominent workers as Meyling (1953)[5] and M. R. Miller (1970)[6] failed to demonstrate the existence of cardiac nerve endings in the human.

The present study is morphological and is an attempt to present what we believe to be the first good photographs of nerve endings in the hearts of the human.

Materials and Methods

The hearts of 21 human adults and infants were studied by means of methylene blue perfusion and immersion. In all cases the hearts of only freshly deceased persons were used. Treatment varied from simple coronary artery perfusion with 0.05% methylene blue in saline to simple immersion of whole opened atria or large pieces of ventricle in 0.005% methylene blue in saline adjusted to pH 3.4. In many cases heart tissue was treated first by perfusion, and then dissected pieces of the perfused heart were immersed. Additional immersion solution was occasionally injected with a syringe and small needle beneath the epicardium and endocardium. Following perfusion and/or immersion, various portions of the heart such as the entire opened right or left atrium and selected areas of the ventricles were aired and then fixed in 8% ammonium molybdate. Following fixation the tissues were thoroughly washed with tapwater, dehydrated with alcohol, and finally cleared and studied in benzyl benzoate.

The advantage of tissues prepared for study in this manner is that one may focus through the entire thickness of the atrial walls, or through a several mm-thick portion of the ventricular wall and easily determine the relative disposition of various types of nerve-end formations.

Observations

The sensory nerve endings found in the human heart are complex unencapsulated endings, encapsulated endings, and end-nets (Figs. 1–9).

Complex unencapsulated endings in the heart are referred to in the literature as diffuse or compact. Endings of moderate-sized myelinated fibers which are much branched and occupy a considerable area may be termed diffuse types of complex unencapsulated endings. From moderately large or large myelinated fibers arise more spatially circumscribed endings of varying size and form. These may be termed compact types of complex unencapsulated endings.

Encapsulated endings are compact, circumscribed nerve-end formations that are surrounded by a definite connective tissue capsule.

The end-net is formed by the anastomoses of the branched dendrites of several apparently different myelinated fibers. It is a prominent and constant feature of the entire endocardium. Since free fiber endings seem to be absent from cardiac tissue, the end-net may either represent free fiber endings in the heart or be a sensory element peculiar to vascular tissue.

In our experience the atrial epicardium as compared to the endocardium is only sparsely supplied with sensory nerve-endings. We have observed both diffuse and compact types of complex unencapsulated endings in the substance of the epicardial connective tissue. Occasionally an end net-like formation was seen lying just above the outer surface of the myocardium. In our series of 21 human hearts we found not even 1 specimen to be richly supplied with epicardial sensory endings.

In the ventrical epicardium a wide variety of both diffuse and compact types of complex unencapsulated endings were encountered. End-nets were generally absent from the epicardium proper.

With our technics we were unable to demonstrate sensory terminals ending about myocardial muscle fibers.

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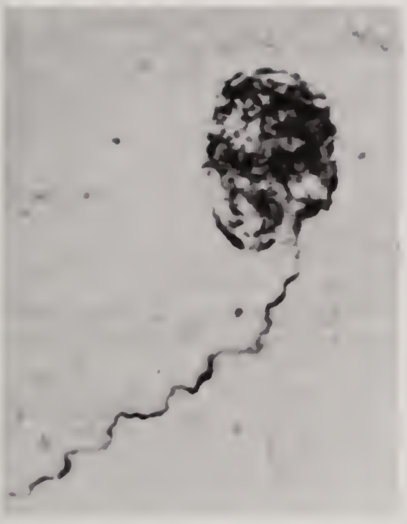

FIG. 1. Normal human infant. Encapsulated ending.

FIG. 2. Human infant in congestive heart failure. A compact complex unencapsulated ending.

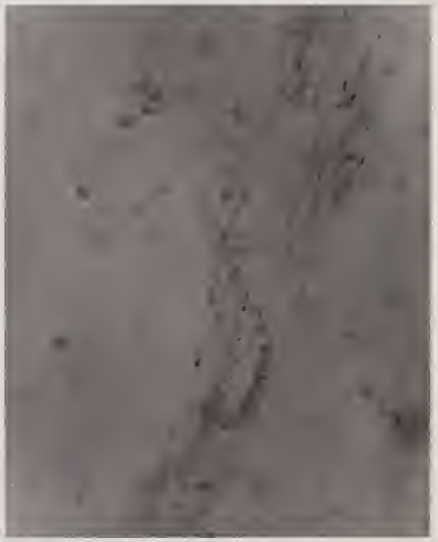

FIG. 3. Human infant in congestive heart failure. Diffuse complex unencapsulated ending.

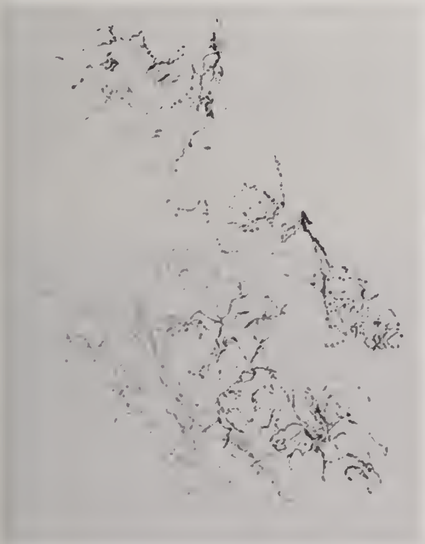

FIG. 4. Infant in congestive heart failure. A spread out but basically compact type of complex unencapsulated ending. (Drawing from original slide).

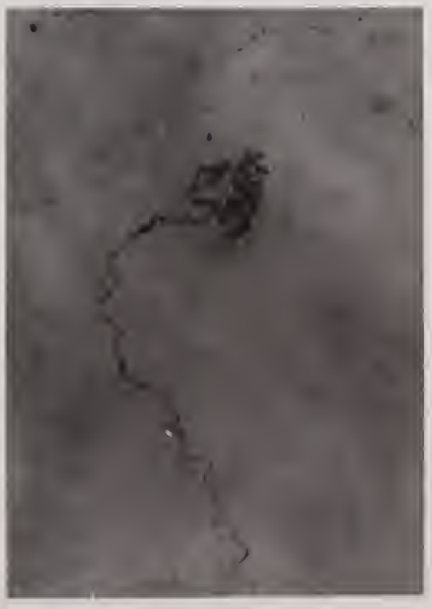

FIG. 5. Human adult, normal. Compact complex unencapsulated ending.

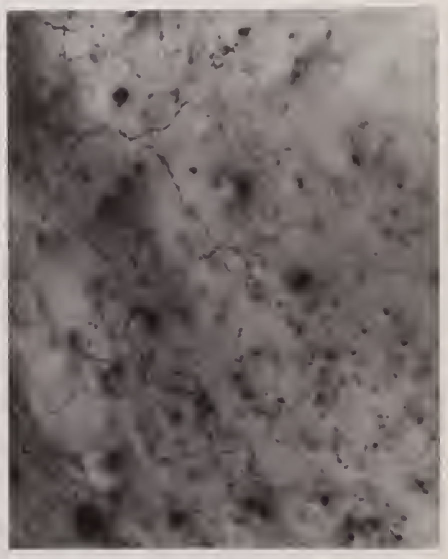

FIG. 6. Human adult. An end-net sensory terminal.

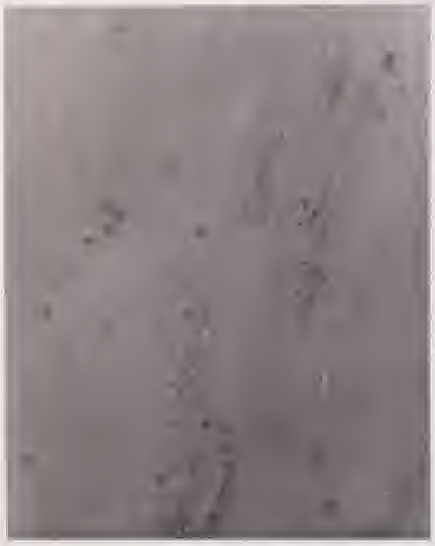

FIG. 7. Human adult. Diseased heart. Diffuse complex unencapsulated ending.

The atrial endocardium is richly innervated. End-nets and diffuse and compact varieties of complex unencapsulated sensory endings occur profusely in this region. Certain locations are more consistently well innervated than others; these are the areas about the entrance of the inferior vena cava into the right atrium, the opening of the pulmonary veins into the left atrium, and the bases of the interatrial septum.

All 3 varieties of nerve endings, end-net, diffuse and compact, may occur in the same location, or each type may occur separately. The end-nets frequently arise from variously-sized myelinated fibers. It appeared to us that the end-net structure arose from the anastomosed dendrites of several nerve fibers. As far as one is able to determine with the light microscope, the branching ends of several fibers anastomose with one another.

The diffuse type of endings arise from fibers approximately 4 to 6 μ in diameter and the branches and ramifications of 1 fiber may occupy an area of several square mm. There are no anastomoses between the terminal branches of different nerve fibers.

The ventricular endocardium is much different from the atrial in that only end-nets and no other types of sensory endings have been observed. In the ventricular endocardium there is a rich nervous plexus. Many of the myelinated fibers in this plexus give rise to sensory end-nets. No particular association of sensory end-nets with the Purkinje bundles was noted.

FIG. 8. Human adult. Diseased heart. Diffuse complex unencapsulated ending.

Discussion

Our studies have led us to believe that both the epicardium and endocardium are well supplied with sensory nerve endings. The majority of the sensory endings in the epicardium are complex unencapsulated in nature. These are remarkably similar in structure to the same type of endings that occur in the dermis, fasciae, aponeurosis, tendons, and periosteum. In the heart, as in other tissues, these endings are probably associated with change in tissue position or a change in the degree of tension or stretch existing in the tissue. Such receptors are termed "stretch" receptors in most of the body but are referred to as baroreceptors in the heart.

The atrial endocardium is the most profusely innervated area of the heart. In both diseased and non-diseased hearts we were able to illustrate a large variety of complex unencapsulated terminals varying from diffuse to compact.

The end-net formation so prominent in both the atrial and ventricular endocardium appeared as separate from the diffuse and compact types of endings. The fact that the end-net is derived from myelinated fibers and shows non-specific cholinesterase staining about the network fibers seems indicative of its sensory nature.

Establishment of the true nature and function of the end-net endings and its granules awaits further investigation. Likewise, the nature and disposition of fiber-mediating pain from the heart remains undetermined.

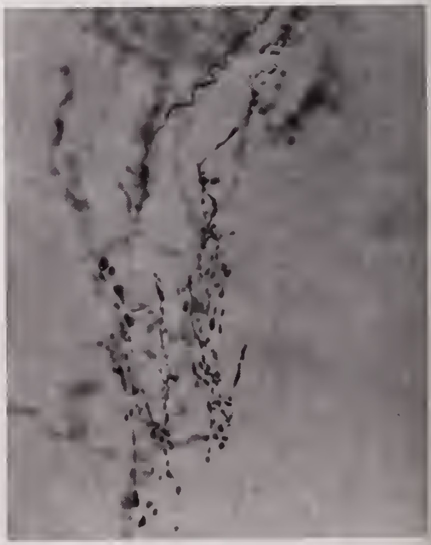

FIG. 9. Human adult, normal. Diffuse complex unencapsulated ending.

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Aspects of Galactose Metabolism in Normal and Galactosemic Cell Cultures[*,†]

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CARL R. MERRIL, M.D.[||] AND MARK R. GEIER[||]

The principal metabolic pathway for galactose involves 3 sequential reactions catalyzed by the kinase, transferase, and epimerase enzymes. The classic deficiency in galactosemia is an absence of transferase enzyme activity although variants do exist.[1,2]

Galactose metabolism has been extensively studied in both normal and galactosemic cells *in vitro*, and a number of *in vivo* investigations have been carried out as well.[3] Since recent work[4] has shown that under appropriate conditions galactosemic cells are able to metabolize galactose to CO_2, the aims of this study were to compare CO_2 production from galactose in normal and galactosemic cultures, and to investigate the utilization of galactose at different substrate concentrations.

Materials and Methods

Five human cell lines were studied: Led-130;[5] WI-38;[6] KB;[7] CCL-72;[8] and JDU.[9] The first 3 are normal with respect to galactose metabolism, while CCL-72 and JDU are human fibroblast lines derived from skin biopsies of 2 documented homozygous galactosemic patients.

Led-130 and KB cells were supplied by Miss Roslyn Wallace; WI-38 cells were obtained from HEM Research, Inc., Rockville, Maryland; CCL-72 cells were obtained from the American Type Culture Collection, Rockville, Maryland; and cell line JDU was a gift of Dr. R. S. Krooth.

Replicate cultures of each cell-line were allowed to grow to confluency in medium MAB 87/3[10] supplemented with 5% fetal bovine serum and penicillin (100 μ/ml). After 3 rinses with phosphate buffered saline, modified medium MAB 87/3 containing galactose-1-^{14}C (Gal-1-^{14}C) (New England Nuclear; 48.1 mCi/mM) but without serum, glucose, or antibiotics was added to each culture vessel which was then sealed with a rubber stopper and incubated at 37° C. At successive intervals, 1.0 cc of gas was withdrawn from each culture vessel by a needle puncture, and the $^{14}CO_2$ in the sample of gas was adsorbed onto a phenethylamine chromosorb column for liquid scintillation counting.[11]

The points on each curve represent an average value of the CO_2 production determinations from each of 2 cell cultures. Each cell line was investigated with galactose concentrations of 10 mμmole/ml and 20 mμmole/ml. The effect of glucose on galactose utilization was also studied and will be reported elsewhere.[4]

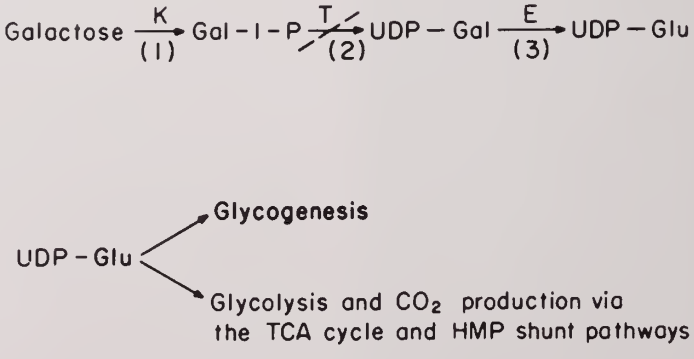
Galactose
K
(1)
Gal-1-P
T
(2)
UDP-Gal
E
(3)
UDP-Glu
UDP-Glu
Glycogenesis
Glycolysis and CO₂ production via the TCA cycle and HMP shunt pathways

FIG. 1. Normal galactose metabolism illustrating the 3 essential enzymatic reactions which permit eventual oxidation to CO_2: (1) K: galactokinase; (2) T: galactose-1-phosphate uridyl transferase; and (3) E: uridine diphosphogalactose epimerase. Abbreviations: Gal-1-P (galactose-1-phosphate); UDP-Gal (uridine diphosphogalactose); UDP-Glu (uridine diphosphoglucose).

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[*] Conducted in the COSTEP Program of the U. S. Public Health Service, Laboratory of Pathology, Division of Biologics Standards, National Institutes of Health, Bethesda, Maryland.

[†] Presented at the annual Student Research Day, George Washington University School of Medicine, February, 1971.

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The spent media from the cell cultures in the experiments reported here were tested in thioglycollate and Sabouraud's media and were sterile; the cells also were periodically shown to be free of mycoplasma. The purity of the Gal-1-^{14}C was confirmed by thin-layer chromatography.

Results and Discussion

The normal metabolic pathways for galactose which lead to CO_2 production are outlined in figure 1. In galactosemic cells having the transferase activity deficit there is a block in the conversion of Gal-1-P to UDP-Gal which, in the absence of alternate pathways, would result in an accumulation of Gal-1-P and no net conversion of Gal-1-^{14}C to $^{14}CO_2$.

$^{14}CO_2$ production as a function of incubation time for the normal cell line Led-130 and the galactosemic cell line CCL-72, each at 2 concentrations of galactose, is shown in figure 2. The data confirm earlier results that galactosemic cells can utilize galactose to a significant degree.[4] Note, however, that there is a relative lag period in the initial hours of incubation and less $^{14}CO_2$ production for CCL-72 as compared with Led-130.

The data for the normal cell-line, LED-130, are representative of the pattern of all 3 non-galactosemic cell-lines; results similar to those for CCL-72 were obtained with JDU.

In both normal and galactosemic cells the amount of galactose converted to CO_2 is a function of the initial concentration of galactose in the medium. Twice as much galactose produces about twice as much $^{14}CO_2$ in normal cells. In the galactosemic cells, on the other hand, the same substrate dependency phenomenom exists but to a lesser degree.

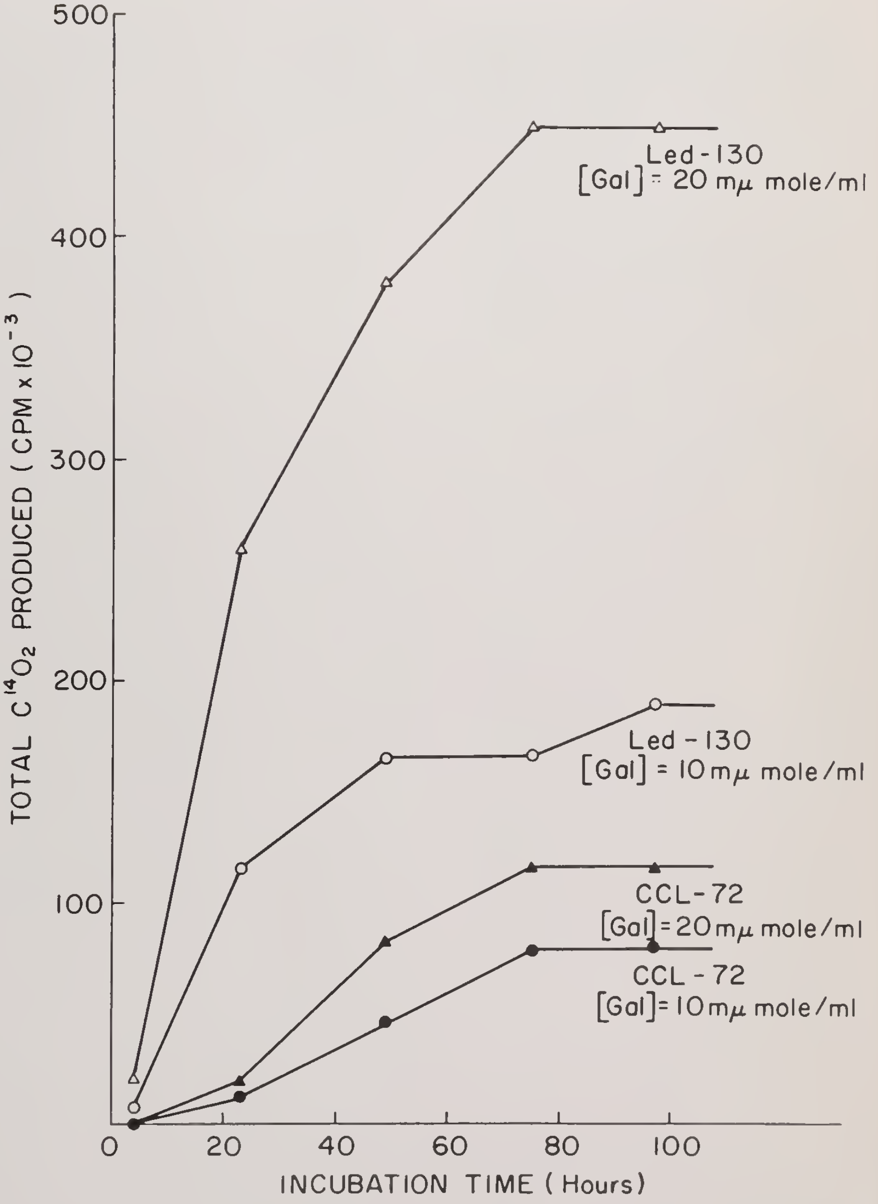

FIG. 2. CO_2 production as a function of time in normal cell-line Led-130 and galactosemic cell-line CCL-72 at 2 concentrations of galactose.

The data in this study suggest that the ability to differentiate galactosemic cells from normal cells on the basis of their inability to convert galactose to CO_2[9] is still clinically meaningful if short (1-2 hours) incubation times are used, in which case the galactosemic cells are in a lag phase and thus act as if they are unable to metabolize galactose. The effect of substrate concentration on the rate and total amount of CO_2 produced implies that the concentration of unlabeled serum galactose must be considered before results of $^{14}CO_2$ screening tests[12] for transferase activity can be validly compared.

Summary

This report has confirmed that galactosemic cells in culture can utilize galactose to a significant degree through alternate metabolic pathways which still lead to CO_2 production, but that the rate and extent of utilization is lower than in normal cells; and that galactose utilization in both normal and galactosemic cells is a function of the extracellular galactose concentration.

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Immunochemical Study of Fetal Cord Sera: Detection of Alpha and Beta Fetal Serum Proteins Associated with Cancer*†

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AND CHAO H. CHAN, M.D.‖

Over 30 years ago Hirszfeld, Halber and Rosenblat[1] showed that an antigenic relationship existed between human tumor extracts and fetal tissue extracts. But only in recent years has the importance of this observation been realized. The finding of an embryonic antigen (CEA) associated with carcinomas of the digestive tract by Gold and Freedman[2] and the alpha-fetoprotein (AFP) detected in sera from patients with primary hepatoma by Tatarinov[3] gives more recent evidence for the embryonic nature of human tumor-associated antigens. It has been postulated that the genes dictating production of these fetal globulins are repressed at birth. In malignant disease these quiescent genes become de-repressed and fetal antigens are again produced.[2]

Several large studies have established the usefulness of AFP detection as a diagnostic test in patients suspected of having primary hepatoma.[4-6] Although Gold's laboratory reported the detection of CEA in 100% of gastrointestinal malignant tumors in a double-blind study[7], the specificity of CEA still remains to be confirmed by other laboratories, but the presence and concentration of CEA in the serum of cancer patients appears to have considerable diagnostic and prognostic value.[8]

We have recently shown that by using highly sensitive pore-gradient acrylamide-gel electrophoretic techniques that several unique serum proteins were detected in cancer-patient sera that were absent from normal sera and non-cancer sera.[9-12] It is now one of our major objectives, aside from determining the physiochemical and biological properties of these unique cancer serum components, to determine their identity. Are these serum components related to fetal serum components? In an attempt to answer this question, it would be necessary to first detect and identify the human fetal serum proteins and to prepare specific immunologic reagents, namely monospecific anti-fetal antiserum. This report describes the detection of alpha and beta tumor-associated fetal proteins with monospecific anti-fetal cord serum antisera.

Materials and Methods

Serum specimens. Individual and pooled newborn cord sera and sera from pregnant women were supplied by Mr. Don Neugebauer, Columbia Hospital, Washington D.C. Sera from patients with primary hepatoma and the various control sera were obtained from area hospitals. The sera was used immediately or stored at −80° C until needed.

Preparation of antisera. Antisera were prepared by hyperimmunization of albino rabbits with pooled fetal cord sera. The cord sera used to prepare the inoculum were first concentrated to 50% of their original volume by lyophilization. Two groups of rabbits were used. Each rabbit of the first group received 2 ml of concentrated cord sera completely emulsified in 4 ml of Freund's complete adjuvant. This mixture was injected intramuscularly (IM), 3 ml into each flank. At 28 and 42 days following the intial injection, subcutaneous (SC) booster injections of 0.6 ml alum-precipitated fetal cord sera was given. Each rabbit of the second group was injected IM with 6 ml of alum-precipitated cord serum. At 14 days the rabbits were injected with 2 ml of alum-precipitated cord sera. A SC booster of 2 ml (0.2 gm) unprecipitated cord serum was given 24 days following the intial injection. Both groups of rabbits were bled by cardiac puncture following the last SC booster injection. The serum was collected in a conventional manner and stored at −80° C.

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† Presented at the annual Student Research Day, George Washington University School of Medicine, February 1971.

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Immunoelectrophoresis and immunodiffusion. Immunoelectrophoresis (IME) was performed with slight modification of the macromethod described by Grabar and Williams.[13] Agarose was used in place of agar as the stabilizing medium. The Ouchterlony macro-gel double-diffusion technique was used.[14]

The rabbit anti-fetal cord serum-antiserum was absorbed with pooled normal human adult serum (NHS) in order to remove antibodies directed against serum proteins common to fetal and adult serum. Immunoabsorption was carried out by adding varying volumes of NHS to a constant volume of antiserum. Absorption was carried out at 37° C for 2 hours and at 10° C for 12 hours. The precipitate resulting from absorption was removed by centrifugation at 3,500 G for 20 min., and the resulting antiserum was tested by immunodiffusion and IME against cord sera (CS), sera from patients with primary hepatoma (HS), and NHS.

Table 1 shows that by using differing volumes of NHS, antisera could be made specific to 3 fetal serum components. Figure 1 shows the IME patterns when either CS or HS was reacted with the specific absorbed antisera. One precipitin arc was found in the region of the alpha-1 globulins, 1 arc in the region of the alpha-2 globulins, and another in the beta globulin region. Antiserum obtained from rabbits receiving CS plus adjuvant and absorbed with 10% (v/v; 0.1 ml NHS/1 ml antiserum) NHS resulted in antibodies specifically directed against the alpha-2 and beta fetal globulins. The alpha-2 globulin was always detected as a weak reaction and found only in a few CS and HS samples. In all the samples tested which were positive for the alpha-2 globulin and beta globulin, especially the HS samples, a partial line of identity was observed (Fig. 1C) suggesting that the beta and alpha-2 globulins share at least one common antigenic determinant. Only the beta globulin was detected when this antiserum was absorbed with NHS at concentrations between 20 and 40%, and no precipitin bands were observed at NHS concentrations greater than 40%.

Antiserum from rabbits immunized with alum-precipitated CS and absorbed with either 10 or 20% NHS resulted in the detection of the alpha-1 globulin and the beta globulin. Absorption with 30 or 40% NHS resulted in the detection of only the alpha-1 globulin; and no precipitin bands were observed at NHS concentrations greater than 40%. It was not possible with any of the alum-CS absorbed antisera to detect the alpha-2 globulin. Antisera monospecific for the alpha-1 fetal globulin was compared with a

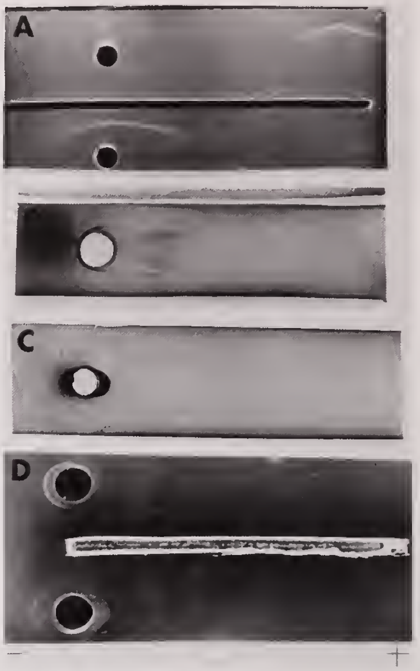

FIG. 1. Immuno-electrophoretic patterns of fetal antigens obtained when either fetal cord serum (CS) or serum from patients with primary hepatoma (HS) is placed in the well, subjected to electrophoresis, and then reacted with absorbed anti-fetal cord serum antiserum placed in the trough. A. Detection of an alpha-1 fetal globulin (upper) and a beta globulin (lower) in CS. B. Beta fetal globulin detected in HS serum. C. Alpha-2 fetal globulin detected in HS serum. Note a line of partial identity with the beta globulin. D. Detection of both the alpha-1 and beta globulins in HS (upper) and their absence in normal human adult serum (lower).

TABLE 1. Specificity of Anti-Fetal Cord-Serum Antiserum after Absorption with Different Concentrations of Pooled Normal Human Serum (NHS)

	Antiserum*					
	Adjuvant			Alum		
Per cent NHS (v/v)	α_1	α_2	β	α_1	α_2	β
5	C	C	C	C	C	C
10	–	+	+	+	–	+
20	–	–	+	+	–	+
30	–	–	±	+	–	–
40	–	–	±	±	–	–
50	–	–	–	–	–	–
↓	↓	↓	↓	↓	↓	↓
100	–	–	–	–	–	–

* Rabbits were either injected with adjuvant plus fetal cord sera or alum-precipitated fetal cord sera (see text).

C = non-specific; antibodies to both fetal and adult serum proteins.

+ = positive precipitin reaction.

– = no precipitin reaction.

commercial antiserum* to alpha-fetoprotein (AFP) by immunodiffusion analysis. Figure 2 shows that a line of immunologic identity is seen, indicating that the antisera prepared in this study were monospecifically directed against AFP.

It was determined that the failure to detect any of the fetal antigens with antisera which had been absorbed with high concentrations of NHS (50-100%) was a factor of dilution and not a result of removal of antibodies directed against the fetal proteins by excess antigen. This was determined by concentrating the absorbed antisera to 50% of its original volume by lyophilization and analysis by IME. It was found that the antigens again could be detected exactly as described above when the antisera were absorbed with 10 or 20% NHS. Similarly, the alpha-1 globulin was not detected with the concentrated adjuvant-CS absorbed antisera or was the alpha-2 globulin detected with the alum-CS absorbed antisera. Absorption at less than 10% NHS resulted in antibodies directed against fetal and adult serum proteins. The concentrated absorbed antisera or the antisera absorbed with 20% NHS were employed in the study presented in the results section.

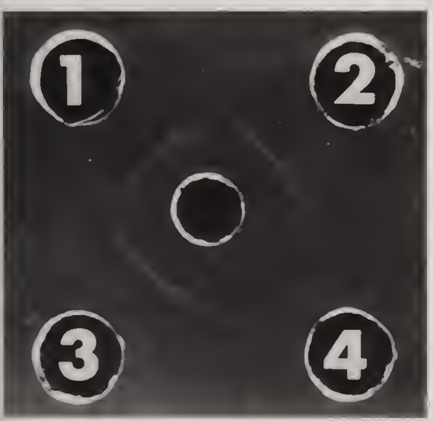

FIG. 2. Immunodiffusion analysis in agar showing a reaction of identity between the precipitin bands resulting when fetal cord serum (center well) is reacted against absorbed anti-fetal cord serum antisera (wells 1 and 2) prepared in this study and a commercial anti-AFP antiserum (well #3). No reactions were observed between these antisera and pooled normal human adult sera (well #4).

Results

Sera obtained from patients with various types of cancer, noncancer disease, pregnant women, and normal individuals were subjected to IME, with the antisera specifically directed against the alpha-1 globulin (AFP) and the beta fetal globulin (BFG) in order to establish the specificity of these fetal proteins. The frequency of these proteins in sera from various sources is presented in table 2. AFP was detected only in the sera from patients with primary hepatoma. BFP was found in 62% of the cancer sera and 13% of sera from pregnant women. Neither AFP or BFP was detected in normal sera. Figure 3 shows that a line of immunologic identity is obtained in agar gel double-diffusion between cancer sera and the serum of pregnant women when reacted against antisera containing antibodies specifically directed against the BFP.

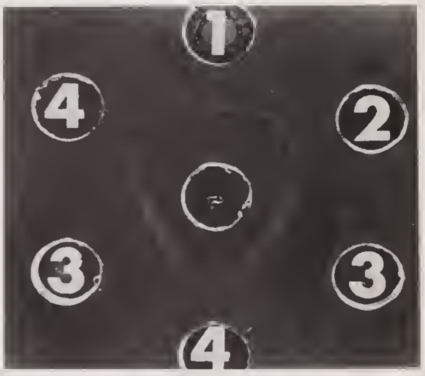

FIG. 3. Immunodiffusion analysis showing a line of identity between the precipitin bands resulting when serum from patients with colon carcinoma (well #1), lymphocytic leukemia (well #2), and serum from a pregnant woman (26 weeks gestation; well #3) were reacted against absorbed anti-fetal cord sera monospecific for the BFP. No reaction was observed between the absorbed antiserum and pooled normal human adult sera (well #4).

Discussion

Our investigation was an attempt to detect fetal protein antigens in fetal cord sera that are associated with malignant disease. With the use of immunoelectrophoresis with absorbed anti-fetal cord serum protein antiserum, 3 proteins with alpha-1, alpha-2, and beta mobilities were detected in fetal cord sera and sera from patients

TABLE 2. Occurrence of Fetal Proteins in Serum.

Serum Source	No.	BFP	AFP
Primary hepatoma	12	11	8*
Alcoholic cirrhosis	8	0	0
Biliary cirrhosis	3	0	0
Carcinoma of colon	9	7	0
Leukemia (various types)	12	4	0
Hodgkin's disease	9	4	0
Tuberculosis	5	0	0
Pregnant women	8	1	0
Normal	25	0	0

*All 12 hepatoma sera were positive when a 2-fold concentration was tested.

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* Fernwood Labs., Inc., Pittsburgh, Pa. 15232

with primary hepatoma. Most importantly, these fetal serum antigens were absent from normal adult sera.

The immunoelectrophoretic analyses of sera from various sources revealed the the alpha-1 fetal globulin was associated only with serum from patients with hepatoma and fetal cord sera. This globulin was subsequently identified as the alphafetoprotein (AFP). The alpha-2 globulin was found in low frequency, and the precipitin reaction was always weak. Because of the weak immunologic reaction, presumably due to the low titer of antibodies directed against this globulin, it was not possible in this limited study to determine if this globulin was tumor associated. The alpha-2 globulin may be the alpha-2H fetal globulin described by Buffe, Rimbaut and Burtin.[15] The alpha-2H was found not to be tumor-specific but probably represents an acute phase-reactant similar to C-reactive protein.

Most interesting was the detection of the BFP in sera from patients with various types of cancer and pregnant women. Most importantly it was not found in normal sera or sera from patients with non-neoplastic disease. The BFP does not appear to be the carcinoembryonic antigen (CEA) described by Gold and Freedman[2] or a similar beta fetal protein isolated from gastrointestinal tumor extracts by vonKleist and Burtin.[16] These beta globulins have a more cathodic mobility and are found almost exclusively associated with carcinomas of the digestive tract. Recently other laboratories have reported that the CEA may be present in sera from patients with other neoplastic diseases.[8]

It is tempting to speculate that the BFP described in our study is the same or similar to the beta fetal globulin described by Takahashi et al[17] or the T-globulin described by Tal and Halperin.[18] Both of these beta globulins, like BFP, are found in sera from patients with various types of malignacies and pregnant women. Takahashi's group used an absorbed antiserum prepared against a hepatoma liver extract. They also detected the beta globulin in cord sera and neonatal sera and, therefore, considered the beta globulin to be of embryonic origin. Although Tal and Halperin reported finding the T-globulin in pregnant sera, no evidence was cited to indicate that this beta globulin is also related to fetal proteins. However, they did not employ antiserum directed against fetal serum proteins but used absorbed antiserum prepared against a crude fraction of adult cancer sera containing the T-globulin.

Even though the number and types of cancer and noncancer patients employed in our study were limited, it appears that both specific (AFP) and non-specific (BFP) tumor-associated fetal antigens can be found in fetal cord sera. A large number of sera from various sources, however, will be required to determine the specificity and uniqueness of BFP. If the specificity of the BFP can be confirmed, it might be of considerable diagnostic value in the subclinical detection of all types of cancer.

It is possible that this unique beta fetal globulin is derived from cells of the liver. The BFP may not be tumor-specific but related to the glycoprotein or symbodies postulated by Apefel and Peters.[19] These glycoproteins are synthesized in the liver in response to stimuli released from neoplastic cells. The liver glycoprotein then attaches to the neoplastic cells, rendering them tolerant to normal cytotoxic immunologic processing and resulting in tumor enhancement. Regardless of the nature and biological mechanisms of BFP, the liver of the hepatoma patient may be the best source for the search for common and specific tumor-associated antigens. Support for this is the report by Krupey, Gold, and Freedman[20] that CEA or a CEA-like molecule could be isolated from primary hepatoma liver extracts.

Presently this laboratory is continuing to search for tumor-associated fetal antigens in fetal sera (obtained at various weeks post-gestation) and hepatoma liver extracts. It is hoped that a rich source of BFP can be found for its subsequent isolation and purification. Once isolated in sufficient quantity, high titer monovalent antiserum can be prepared which would circumvent some of the difficulties encountered with the use of antiserum made specific by immunoabsorption.

Summary

Immuno-electrophoresis, with the use of absorbed anti-fetal cord serum antisera, resulted in the detection of 3 fetal serum globulins: an alpha-1, and alpha-2, and a beta globulin. The alpha-1 was identified as the alphafetoprotein and was found only in newborn cord sera and sera from patients with primary hepatoma. The beta globulin (BFP) was found to be a common fetal tumor-associated antigen, being detected in 62% of the sera from patients with various types of cancer. The diagnostic value and relationship of the BFP to other tumor-associated embryonic antigens were discussed.

Acknowledgments

The authors are indebted to Mrs. Dianna Roberts and Mrs. Karren Farrell for excellent technical assistance.

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Late-Onset Epilepsy*†

HAROLD STEVENS, Ph.D., M.D.‡, AND BRUCE JORGE AMMERMAN§

This study was undertaken to test the validity of the widely accepted rubric that onset of seizures after age 20 is often due to neoplasm. This position is categorically stated by Penfield[1] and emphasized by other eminent authorities.[2,3] Based on extensive experience at the Montreal Neurological Institute, Penfield further stated that trauma is the commonest cause of seizures that begin between the ages of 18 and 35, and added, "in cases of onset of seizures between 35 and 55 the most frequent cause is cerebral neoplasm with cerebral trauma and vascular sclerosis to be considered secondarily." This citation reflects the opinion of many neurologists and neurosurgeons.

This claim needs to be re-examined for both theoretical and practical reasons, particularly since most prior statistical studies on late-onset epilepsy have been done at large prestigious centers where non-representative cases tend to gravitate, thus adding scope to skewness.

In order to obtain a random representative sample, 87 ambulatory patients were studied ranging in ages from 25 to 55. They were divided into 2 groups of 40 consecutive patients from private practice and compared with 47 unselected inpatients at the George Washington University Hospital whose seizures began in the designated age period.

Figure 1 illustrates the age range of the 2 groups. Follow-up ranged from 1 year to 20 years. Distribution of age showed about three-fourths of the private patients were in the 35-55 age-group compared with about two-thirds of the hospital patients. The 1:1 ratio of men to women reflects a relatively higher incidence among women than reported in other series.[4-6]-1,500 non-institutionalized patients Lennox[7] found a ratio of 3 women to 5 men whose first seizure began after the age of 20. The disparity between our experience and that of others might reflect the different population from which our data were drawn, that is, a non-industrial population, where the role of trauma may be less than average and hence not reflect the higher incidence of trauma to which men are exposed. However, several reports in the literature, particularly in more recent years, are consonant with our findings.[8-13] The total incidence of cases with traumatic epilepsy was low in contrast to other series, e.g., in a report by Serafetinides and others[14], out of 51 patients over 25 years of age who had their first seizure, 17 gave a history of head trauma of which 7 were severely hurt.

In addition to trauma, several other categories of causation were identified,

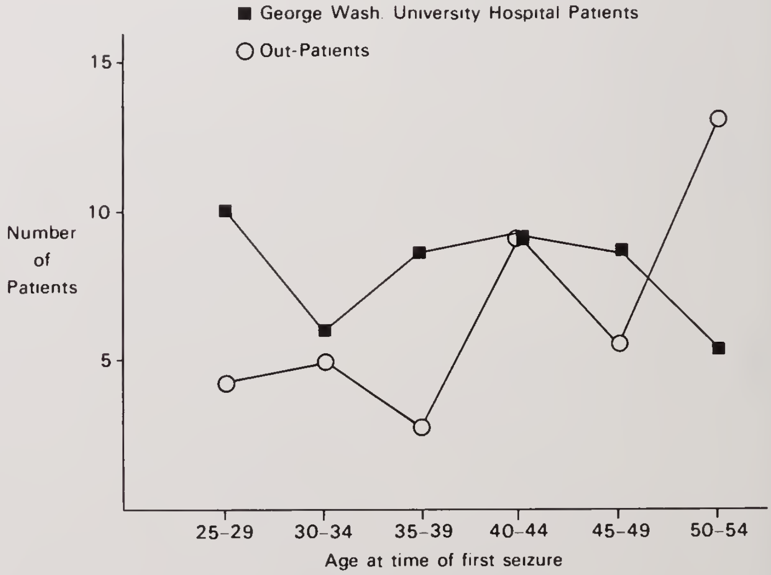

FIG. 1. Age range between hospitalized and private patients. There is a significant difference between the two populations at p value $< .05$.

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* Presented at the annual Student Research Day, George Washington University School of Medicine, February 1971.

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(fig. 2). Only 8 patients had brain tumors. This low incidence is in contrast to other reports[2,3,15,16] and particularly that of Penfield.[1]

The discrepancy between our data and prior reports might reflect the inadvertant selection of patients reported from large medical centers, chronic hospitals, or other institutions. It is our belief that data on such samples are not necessarily representative of ambulatory patients seen in private practice in the Washington metropolitan area. Six of the 8 cases of brain tumors were in the hospitalized group. Brain tumor often signals its presence first by a convulsion,[17] the frequency of this phenomenon varying between 0 and 45% of adult patients with brain tumor having seizures.[6,9,15,16,18] Fifteen % have seizures as their sole initial symptom. (Walker, 1936[19]). Of 92 patients with seizures beginning after the age of 60, 22% had brain tumors and 40% suffered a seizure as the initial symptom.[20]

In addition to the relatively low incidence of brain tumors as causative of late-onset seizures, our data also tend to controvert the claim that late-onset seizures have a poor prognosis, regardless of causation.

Seventy-six % of our patients not only had no evidence of tumor but no known progressive pathologic process. In 26% only 1 seizure was suffered, all were grand mal. Thirty-six % had 3 or fewer seizures. Neurologic examination was negative in 70%. Further, in contrast to other reports, the preponderance of this group, that is, 41 cases, were eventually diagnosed as idiopathic.

Several interesting and unusual mechanisms were encountered, for example, 2 cases were associated with hyperostosis frontalis interna, sometimes labeled metabolic craniopathy. The exact importance of hyperostosis interna is a matter of dispute, but most consider it benign and irrelevant.[21]

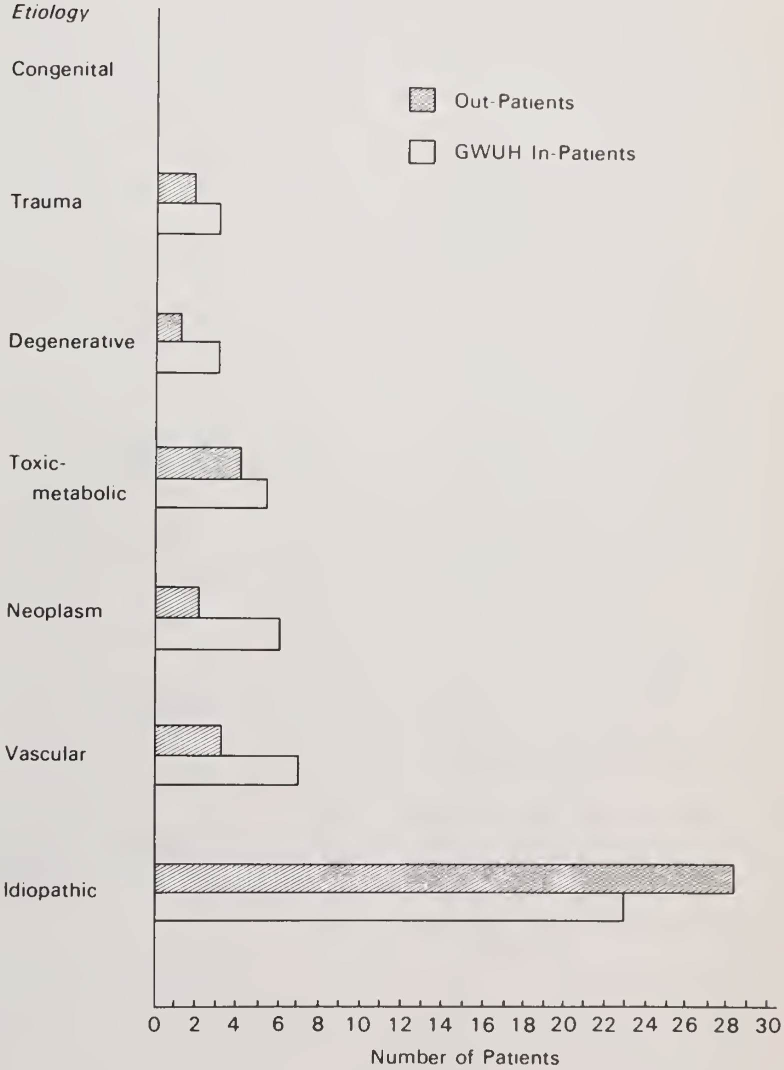

FIG. 2. Comparison between number of private outpatients and hospitalized patients in six etiological categories.

One case of reading epilepsy has been followed with complete control of seizures on Dilantin and with avoidance of prolonged reading.[22] One instance of puerperal hemiplegia was found with complete restitution of function and no sequalae after 12 years of follow-up.[23]

A single instance of allergic epilepsy, a rare entity,[24] was included in this series: A 32-year-old woman who had no prior neurological disease or seizures was given a routine tuberculin skin test. Within 3 minutes she had a generalized seizure with incontinence which lasted about 15 minutes. Neurological examination was negative, the EEG was normal and has remained so over an 18-year period of follow-up. No further seizures occurred, but transient attacks of erythematous eruptions and urticaria occurred occasionally.

None suffered status epilepticus, an entity which always implies biochemical or structural brain defect and carries a poor prognosis if the onset is in an adult.[25]

Electroencephalograms were made in 72 of the patients; 61% were abnormal (fig. 3). Of those with single seizures, the incidence of abnormal EEGs was quite low, less than 35%, consistent with the experience of others.[4,5,6,12,14,26]

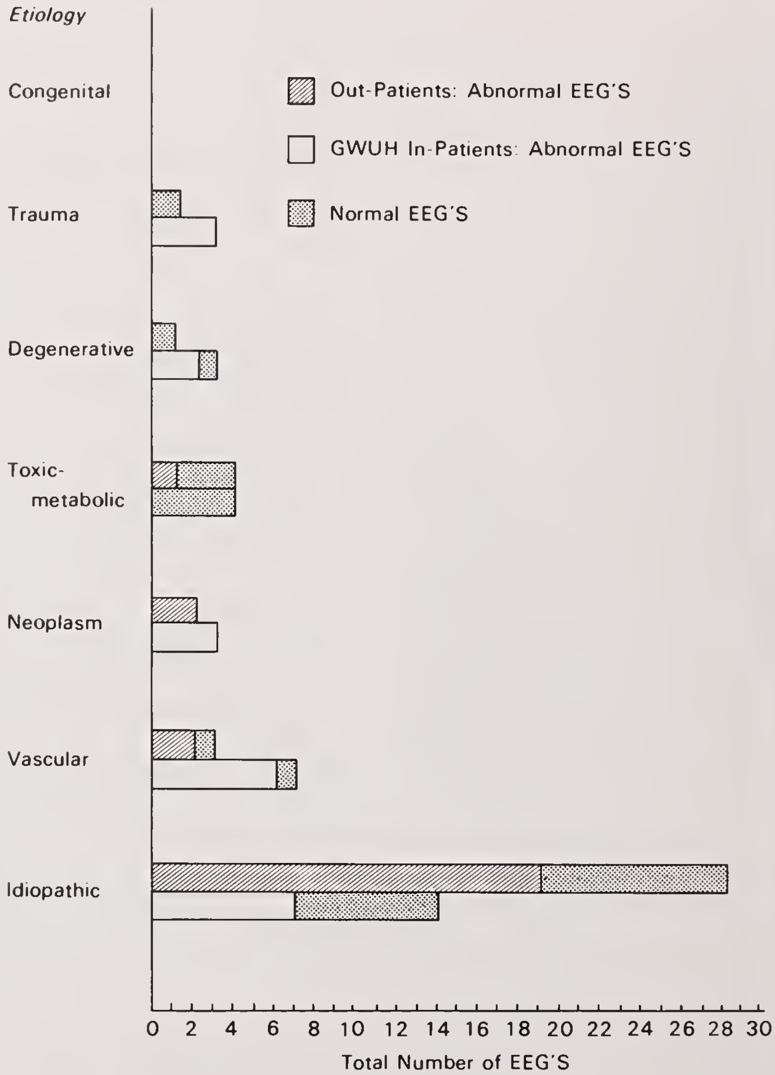

FIG. 3. Distribution of abnormal EEGs in the six etiological categories in the two populations.

Of 9 patients with psychomotor epilepsy, all had abnormal EEGs, 5 of which were due to brain tumors and demonstrated focal EEG abnormalities. The EEG correlated closely with the clinical type of seizure, as reported by Raynor et al.[12] In the series reported by Carney and others[20] 20% of patients with brain tumor had normal EEGs; 24% of non-tumor patients had normal EEGs. Focal delta activity was the most frequent abnormality in patients with brain tumor. Similar EEG correlations in late-onset epilepsy were reported by Takahashi[27] and Kawaguchi.[6] The latter found brain tumor as the cause of seizures after the age of 20 in only 6 patients out of 207. They also observed that in all patients with a poor response to medication, the EEG was abnormal with a high incidence of focal discharges.

In summary, the current acceptance of a sinister prognosis in late-onset epilepsy is disputed by the data presented. In unselected ambulatory patients in private practice the prognosis is excellent. Brain tumor and trauma are found to be relatively uncommon causes, and the preponderance of cases will eventually be labeled idiopathic. A large percentage will have single seizures; thus, most patients with the onset of a seizure after the age of 25 can be reassured and can be spared the pain and risks of pneumoencephalography and arteriography, particularly if the neurological examination is negative, the EEG is normal, and the seizures are generalized rather than focal. Data from these 3 sources are particularly helpful in deciding the advisability of permitting the patient to drive a motor vehicle.

Needless to say, a seizure should never be dismissed as trifling; careful examination and follow-up is mandatory.[28] With EEG and brain scan techniques most patients can be followed without risk of overlooking a treatable lesion.

Acknowledgements

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Ultrastructure of the Intima of the Central Retinal Artery[*†]

MAXWELL A. HELFGOTT[‡]

The central retinal artery of the optic nerve is a small muscular branch of the ophthalmic artery which supplies the retinal arterioles which are observed ophthalmoscopically. The purpose of this study was to examine by light and electron microscopy the structure of the central retinal artery, with special reference to its intima. Although the structure of the optic nerve and its components has recently been reviewed by Anderson,[1] it was thought that a more detailed examination of this crucial vessel by EM would not only provide a suitable subject of investigation as a parttime summer project while learning EM technical preparation and processing with Dr. Ben S. Fine at the Ophthalmic Pathology Branch, Armed Forces Institute of Pathology, but might also provide some more up to date information on a subject that has received little attention in the past decade.

A young, healthy Rhesus monkey was anesthetised and perfused intraarterially with 4% glutaraldehyde. Small blocks of tissue were removed from the anterior part of the intraorbital optic nerve near the sclera. The blocks were rinsed in phosphate buffer and placed in Dalton's chrome-osmium tetroxide fixative for 1 hour. They were dehydrated in a series of ethanol baths, infiltrated in mixtures of propylene oxide and epon, and embedded in capsules with pure epon.

One-micron thick sections were mounted on glass slides and stained with phenylene diamine. Thin sections were cut on an automatic microtome and stained with uranyl acetate and lead citrate.

Results

Light microscopy revealed a typical small muscular artery surrounded by a fibrous adventitia. The media appeared to be composed of 4 to 6 layers of smooth muscle cells. The next layer distinguishable with the p-phenylene diamine stain was the internal elastic lamina, which appeared discontinuous throughout its circumference. Internal to that was the intima and subintimal space. In various arcs of the intima, cells were noted in the subintimal space.

Electron microscopy showed the media of the artery to be composed exclusively of smooth muscle cells[2] in support of the multifunctional concept of smooth muscle.[3]

The internal elastic lamina was seen as an electroluscent, stippled ring beneath the media. It was noted to have numerous attenuations or fenestrations typical of muscular arteries. Quite frequently, projections from the medial smooth muscle were seen to pass through these openings into the subintimal space (Fig. 1). Less frequently, endothelial processes would be seen pointing toward the elastica. On one occasion, contact between medial and endothelial processes was seen. The significance of such activity is not certain, although one study[4] indicates that medial muscle may be involved in endothelial replacement, at least in the injured vessel. This may represent a means of transfer of nutrients to the avascular media.[5]

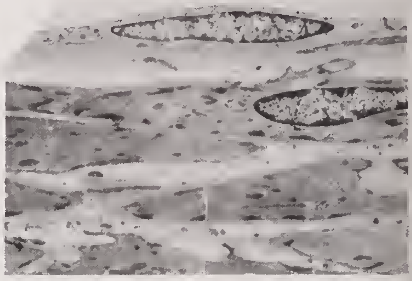

FIG. 1. Intima of central retinal artery. Note smooth muscle process projecting through a break in the internal elastic lamina (light stippled area beneath the endothelial cell) (× 3950).

Another feature of the intima was the presence of elongated cells in fairly extensive and discrete areas of the subintimal space (Fig. 2). They met all of the criteria for smooth muscle, namely, longitudinal filaments attached to plasmalemmal and cytoplasmic dense bodies, pinocytotic vesicles, and a continuous though somewhat diffuse-basement membrane. It was noted that the number of filaments and densities were greatly diminished compared to the medial smooth muscle. In fact, the cells appeared morphologically intermediate, yet distinct from the medial smooth muscle and endothelium.

These smooth muscle cells have

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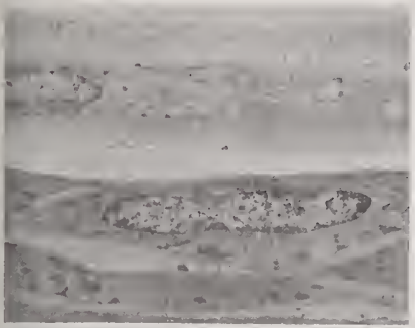

FIG. 2. Note continuous internal elastic lamina and the presence of a layer of cells between it and the endothelium (X 3900)

been previously described in other arteries[6,7] and have attracted a great deal of interest in the field of experimental atherosclerosis[7] and vessel wall repair.[2,4] Buck[2] coined the term myointimal cells. It has been shown that these cells arise from dedifferentiating medial smooth muscle[2,3] and that they greatly increase in experimental atherosclerosis and generally increase in numbers with age.[8]

The relationship of the structure of the central retinal artery to its pathology in this area is unknown. Atherosclerotic disease there is common, and the region of the lamina cribosa is a frequent site of occlusion. Anderson[1] noted that the internal elastic lamina ends abruptly at the lamina cribosa; the factors which contribute to the pathology may involve this transition from internal elastic support to scleral and intra-ocular pressure defence against luminal hydrostatic forces. If there is normally a greater amount of intimal damage at that point, and if the mechanism of repair involves the myointimal cell, it would be logical that those factors that predispose to atherosclerotic disease, which in fact involves these cells or their pathological derivatives, would occur most frequently in the region studied.

These cells have been reported to be seen occasionally in the larger intracranial arteries within the first decade in humans and to increase in numbers with age. In the central retinal artery sections examined, one quarter to one third of the intima was involved, despite lack of evidence of a pathological process. Admittedly, this study is quite limited, but further study into similarly normal and similarly prepared monkeys would be warranted to confirm the findings presented here.

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The Vanishing Diaphragm in Pericardial Effusion

DONALD J. SIPLE, M.D.*†

The dilemma of separating an enlarged heart from a pericardial effusion confronts many cardiologists when evaluating the plain chest radiographs of cardiac patients. When we are able to make a specific diagnosis from plain film studies this is always gratifying but unfortunately infrequent as evidenced by the phenomenal increase in special diagnostic procedures. Plain-film diagnosis at times may avoid unnecessary special diagnostic tests, especially in the very young and the very ill. Plain-film diagnosis may aid in selecting patients more discriminately for special diagnostic techniques. This study is an attempt to make the plain-film diagnosis of pericardial effusion more specific and to separate enlarged hearts from pericardial effusions with plain films of the chest.

Theoretical Considerations

With the gradual accumulation of pericardial fluid the pericardial sac will tolerate up to a liter or more of fluid, often without embarassing the heart and presenting a variety of clinical pictures. With the slowly developed pericardial effusion, especially with underlying heart disease, plain-film diagnosis can play a very important role.

There are a number of plain-film X-ray signs that direct one toward a pericardial effusion:

1. Change in heart size. An increase in heart size on serial roentgenograms over a short period is associated with pericardial effusion.
2. Change in heart shape. As the heart enlarges with pericardial effusion it becomes more globular. This globular shape is characteristic with large effusions, and at least 5 different types have been described as pathognomonic: the triangular, the trapezoid, the spherical, the water bottle, and the onion. The common denominator of all of these is loss of the normal indentures of the heart. There is a disproportionate widening of the heart with pericardial effusion, that is, the transverse diameter increases more rapidly than the cardiac length. The cardiophrenic angles become more acute.
3. Change of the heart with position. With change in position from erect to recumbent, the base of the heart is said to widen due to the presence of pericardial fluid.

Unfortunately, all of these changes in size and shape associated with pericardial effusion may occur with an enlarged, dilated heart. An enlarged heart may also widen with change in position. Finally, many pericardial effusions are atypical and never conform to the characteristic shape that we expect even with massive accumulation of fluid within the pericardial sac. No plain-film technique can distinguish an enlarged heart from an effusion, and we commonly rely on special procedures to make this distinction.

Attempting then to make the plain-film diagnosis of pericardial effusion more specific, changes in the left hemidiaphragm have been studied, explicitly that segment of the diaphragm seen through the heart shadow. This medial intracardiac segment of the left diaphragm is 1 of the most constant features of the normal chest X-ray film (Fig. 1), extending from the medial paravertebral shadow to the left lateral chest wall. A thin, radiolucent band follows the outline of the diaphragm along the upper margin. This accompanying shadow is a radiographic phenomenon. When the diaphragm is diminished or obliterated by disease, this radiolucent line is lost. Anatomical and pathophysiological considerations relate the distribution of pericardial fluid to this segment of the diaphragm.

The fibrous pericardium is fused to the central tendon of the diaphragm (Figs. 2 and 3). Osler in 1914 described inferior displacement of the diaphragm and liver beneath large pericardial effusions, noted at necropsy. Williamson in 1920 demonstrated inferior displacement of the diaphragm and left lobe of the liver in cadavers with agar gel casts of experimental pericardial effusions. He studied the distribution of fluid within the pericardial sac because of his "dissatisfaction with the roentgen ray in diagnosing pericardial effusion." More pericardial fluid is distributed along this diaphragmatic surface than any other segment of the pericardial sac, as Steinberg et al[4] in 1958, and more recently Soulen et al[17], and others have shown (Fig. 4). Soulen and his group, also demonstrated a shift of pericardial fluid toward the diaphragm during inspiration, as the central tendon is drawn downward increasing the volume of the thorax and elongating the pericardial sac. These changes in the cardiac silhouette and the left hemidiaphragm should be reflected on plain X-ray films of the chest made while the patient holds a deep breath.

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† Awarded First Place at the Annual Philip Burka Award presentations, 1971.

Clinical Material

These theoretical considerations relating the distribution of pericardial fluid to the diaphragm led to a review of pericardial effusions. These plain chest radiograms have shown that the left hemidiaphragm within the heart shadow is effaced and often obliterated by the accumulation of pericardial fluid (Figs. 5–25).

At the Georgetown University Hospital many enlarged heart shadows are seen on plain chest radiograms because of very active medical and surgical cardiac services. A review of enlarged hearts, as well as correlation with necropsy findings, heart pool scans, and angiograms to exclude pericardial effusion, has shown that moderate cardiomegaly, either generalized or with specific chamber enlargement, leaves the diaphragm unchanged (Table 1). The left medial segment of the diaphragm remains clearly outlined and may be seen through the heart shadow with moderate increase in heart size (Figs. 26 and 27).

Massive enlargement of specific heart chambers will also leave the left diaphragm unaltered with the single exception of the left ventricle. Rarely, an enormous left ventricle may efface the diaphragm, probably because the left ventricle is the only heart chamber expanding directly into the left hemidiaphragm simulating the accumulation of pericardial fluid. Ewart's sign, which is associated with pericardial effusion at times may be elicited with a prominent left ventricle (Table 2).

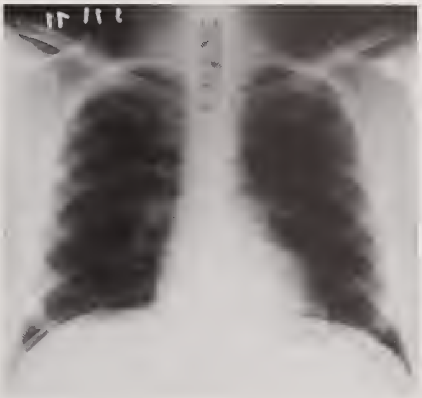

FIG. 1. Normal chest and normal cardiovascular silhouette. Left hemidiaphragm is clearly seen through heart shadow. A thin radiolucent line follows the upper margin of the diaphragm and is a radiographic phenomenon. This segment of diaphragm is diminished and often obliterated by pericardial fluid.

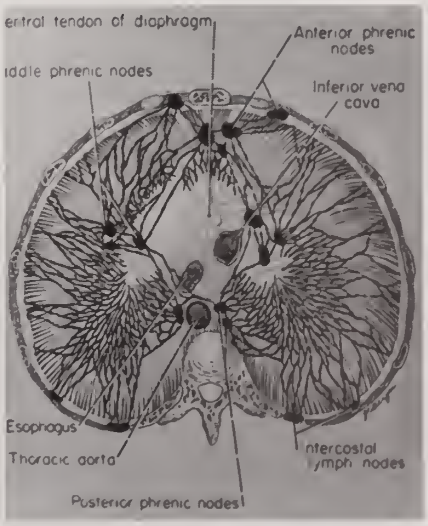

FIG. 2. Diaphragm seen from above, showing position of central tendon. The central tendon lies more to the front than the back of the thorax and occupies more of the left than the right hemidiaphragm. The central tendon corresponds to the anatomical distribution of the diaphragmatic pericardial sac.

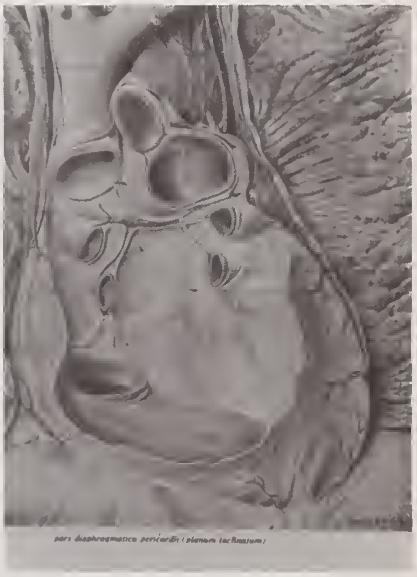

FIG. 3. Pericardial sac with heart removed. The diaphragmatic pericardial sac is demonstrated as seen from the front.

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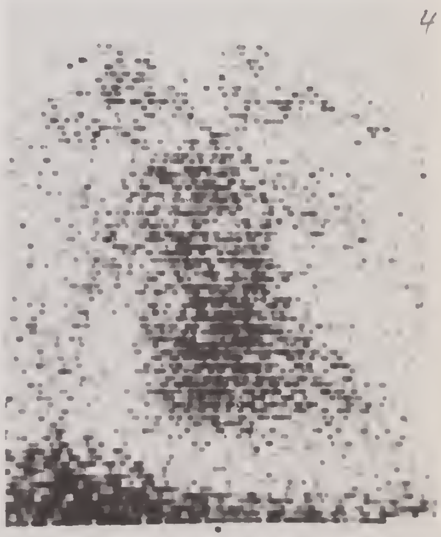


FIG. 4. Heart-pool scan of large pericardial effusion. The conspicuous clear area beneath the heart pool represents a large collection of fluid within the diaphragmatic pericardial sac.

TABLE 1

II 50 Cases Cardiomegaly with L.V. Enlargement

Diagnosis	Cases	Diaphragm Normal	Dim.	Oblit.
A.I.-A.S.	16	9	4	3
Lt. Vent. Aneurysm	3	2		1
M.S.-M.I.	8	6	2	
P.D.A.-Coarct.	15	10	5	
Misc.	8	6	2	
Total:	50	33	13	4

50 patients all proven to have cardiomegaly with enlarged left ventricles. This table correlates changes in the left hemidiaphragm observed on the PA upright observed on the PA upright chest radiogram with the final diagnosis in each case. The left hemidiaphragm was obliterated in 4 and diminished in 13 of these patients without pericardial effusion

TABLE 2

27 Cases Suspected Pericardial Effusion

Diagnosis	Cases	Diaphragm Normal	Dim.	Oblit.
Cardiomegaly	6	6		
Thickened Pericardium without effusion	4	4		
Pericarditis with effusion	3		2	1
Pericardial effusion	14	1	1	12
Total:	27	11	3	13

27 patients with suspected pericardial effusion; the final diagnosis was proven in each case by necropsy, angiography, heart pool scan or surgery. This table correlates changes observed in the diaphragm on the upright PA chest radiogram with final diagnosis in each case. Change in the left hemidiaphragm was observed in 16 or 17 cases of pericardial effusion. In the single case of pericardial effusion with the diaphragm normally outlined on the chest radiogram a loculated effusion may have been present, leaving the diaphragm unaltered. This was not proven.

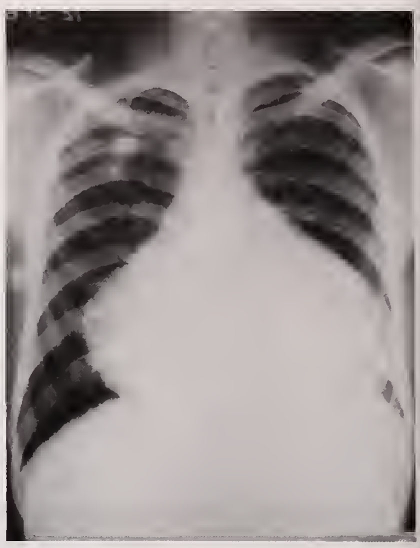


FIG. 5. Case 1. Post-traumatic pericardial effusion following a stab wound. Configuration of the cardiac silhouette is typical of pericardial effusion. The left hemidiaphragm has been obliterated by the effusion and is not seen through the heart shadow. The lateral view (not shown) demonstrated the left lower lobe to be free of pulmonary disease. The rounded density in the right upper lung field proved to be an artifact.

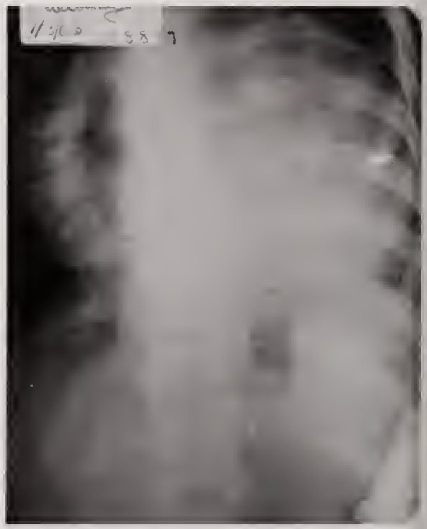


FIG. 6. Case 1. Carbon dioxide angiogram demonstrating the large post traumatic effusion.

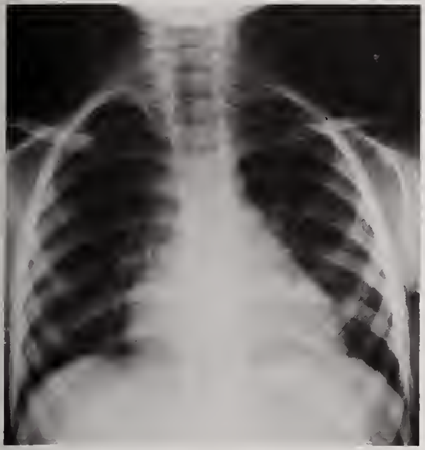


FIG. 7. Case 2. Four-year-old female child with Still's disease. This film was made about 1 year before admission to hospital with pericardial effusion. The left hemidiaphragm is seen through the heart and is normally outlined.

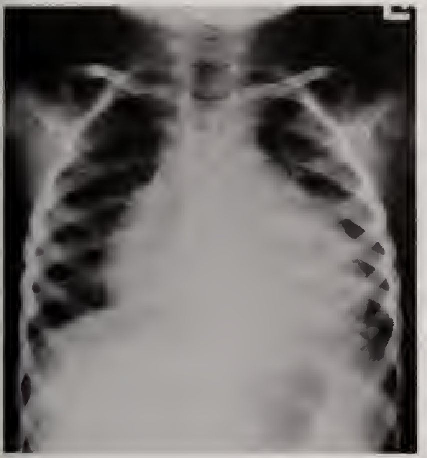


FIG. 8. Case 2. This film was made about 1 year after the film shown in figure 7. The cardiac silhouette is greatly enlarged and a heart-pool scan (not shown) demonstrated a large pericardial effusion. The left hemidiaphragm has been obliterated and is not seen through the heart shadow.

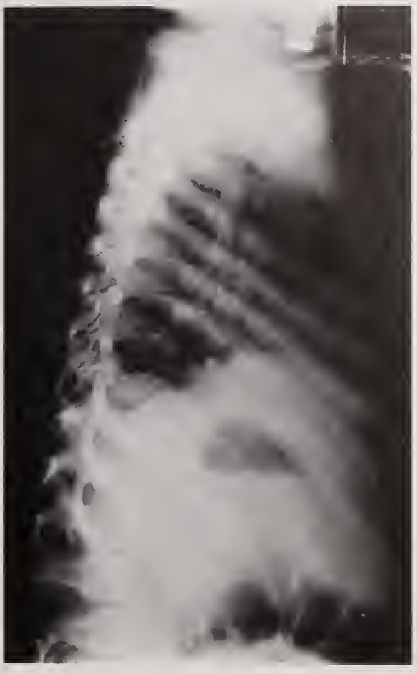


FIG. 9. Case 2. Lateral view of chest shows left lower lobe to be free of pulmonary disease. This study was obtained on the same date as figure 8. Obliteration of the left hemidiaphragm seen on frontal view may be attributed to the large collection of fluid within the pericardial sac.

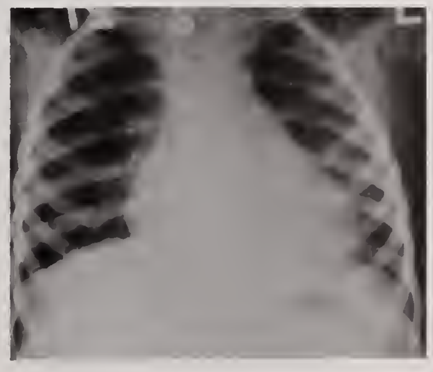


FIG. 10. Case 2. 1 week after films shown in figures 8 and 9, cardiac silhouette is smaller as the patient responds to therapy. The left hemidiaphragm is diminished but is beginning to re-appear.

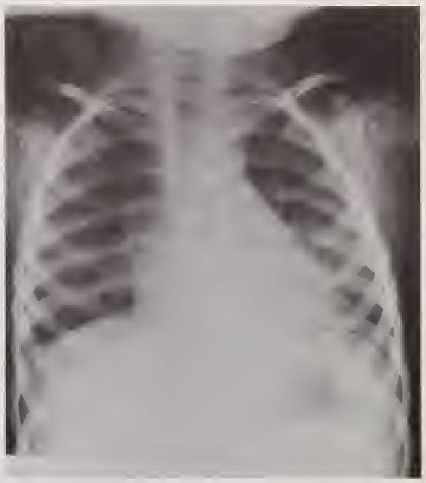


FIG. 11. Case 2. Approximately 1 month after onset of symptoms the heart has resumed its normal configuration. The left hemidiaphragm is again clearly outlined through the heart shadow following resolution of pericardial fluid.

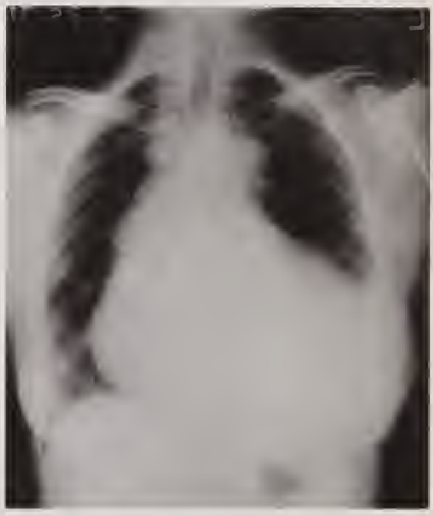


FIG. 12. Case 3. A 62-year-old woman admitted to the cardiac surgical service for evaluation. On this upright radiogram the cardiac silhouette is not the typical water bottle configuration that is associated with pericardial effusion, but the left hemidiaphragm is not seen through the heart shadow.

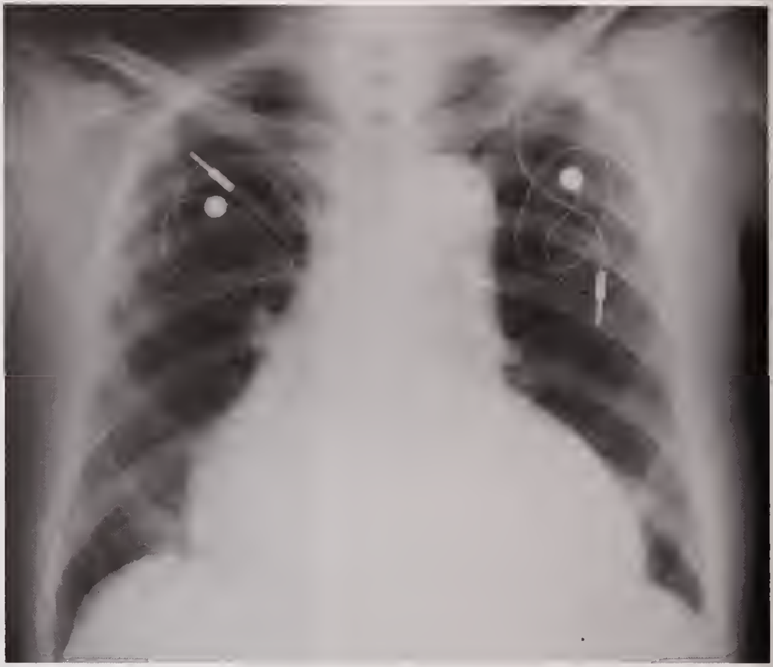


FIG. 14. Case 4. A 56-year-old man admitted for myocardial infarction developed changing arrhythmias and a pericardial friction rub. Although the cardiac silhouette was not typical, a pericardial effusion was suspected clinically. The left hemidiaphragm is not seen through the heart shadow and has been obliterated. The lateral chest radiogram (not shown) demonstrated the left lower lobe to be free of disease.

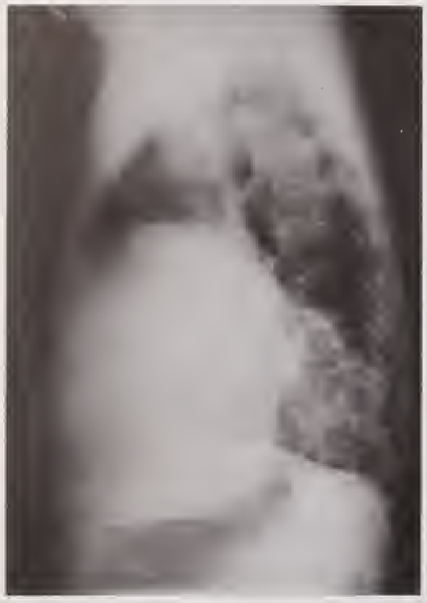


FIG. 13. Case 3. Lateral chest radiogram shows left lower lobe to be free of pulmonary disease or pleural fluid. The rounded density in the retrocardiac area, projected over the spine, was thought to be a bulging postero-lateral pericardial sac. Obliteration of diaphragm was thought to be due to a pericardial effusion. A heart-pool scan demonstrated a large collection of pericardial fluid.

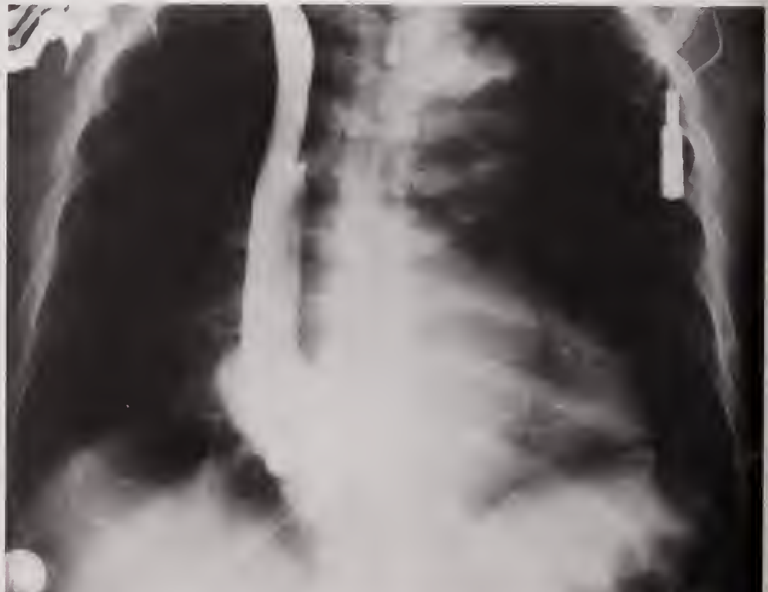


FIG. 15. Case 4. Venous angiogram demonstrates a large pericardial effusion.

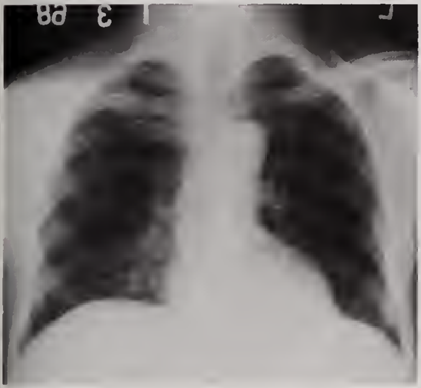


FIG. 16. Case 5. In a 42-year-old man with chronic uremia the heart shows left ventricular prominence. The left hemidiaphragm is clearly outlined through the heart shadow.

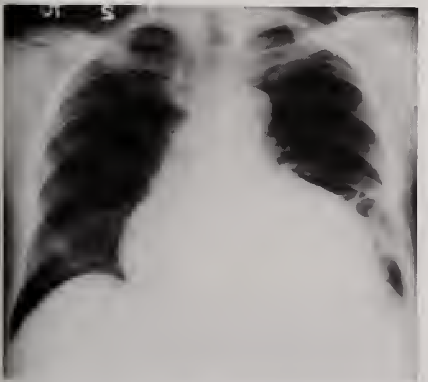


FIG. 17. Case 5. 2 years later the heart silhouette has significantly increased in size with a globular configuration. A pericardial effusion was suspected and demonstrated by a heart-pool scan (not shown). The left hemidiaphragm is not seen through the heart shadow.

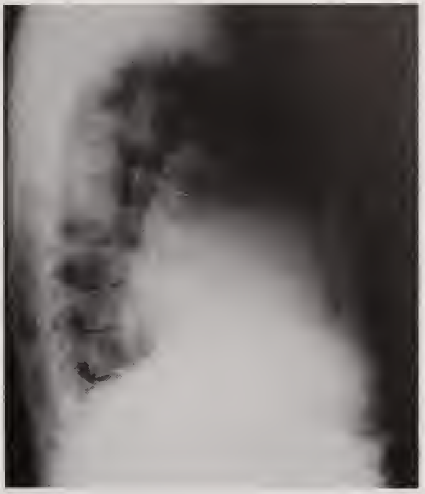


FIG. 18. Case 5. Lateral view demonstrates the left lower lobe to be free of pulmonary disease. Obliterations of left hemidiaphragm is attributed to pericardial fluid.

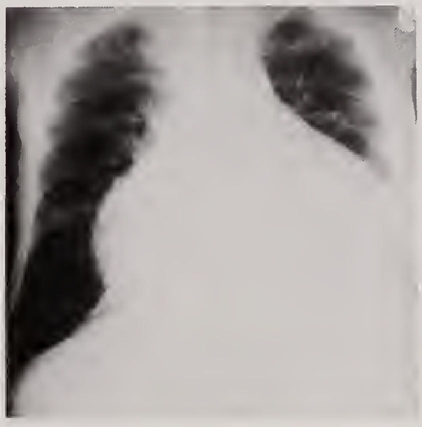


FIG. 19. Case 6. Large typical pericardial effusion in a 62-year-old chronic uremic that has obliterated the left hemidiaphragm. The pericardial effusion was demonstrated at necropsy. The left lower lobe was free of disease.

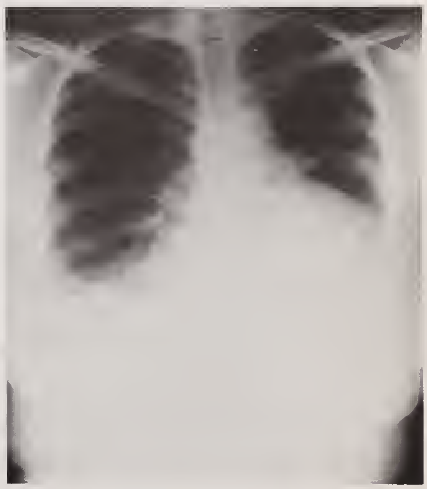


FIG. 20. Case 7. (atypical). A 46-year-old woman with left chest pain was suspected to have pulmonary embolism. A lung scan showed normal perfusion of the lungs. The cardiac silhouette is greatly enlarged and the left hemidiaphragm is obliterated. A pericardial effusion was suspected.

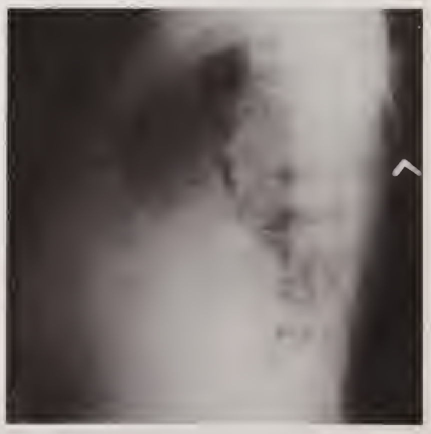
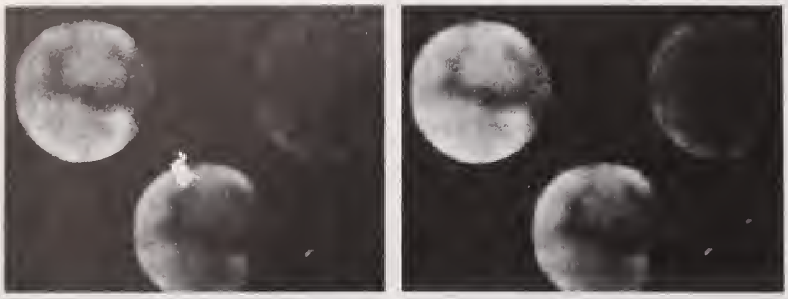


FIG. 21. Case 7. Lateral view of chest shows left lower lobe to be clear.



FIGS. 22. Case 7. Heart-pool scan demonstrates a large pericardial effusion.

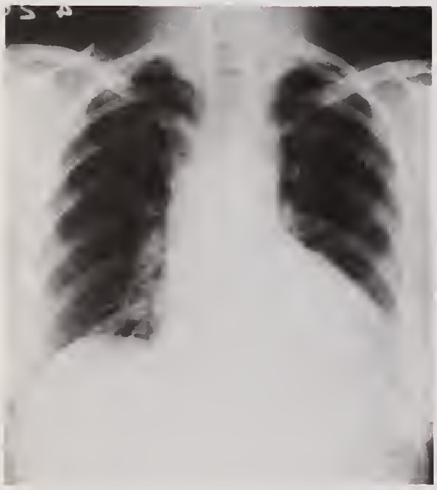


FIG. 23. Case 8. 47-year-old woman with cardiomegaly and left ventricular enlargement from cardiac disease, possibly myocardopathy. The left hemidiaphragm is normally outlined. No radiographic or clinical sign of pericardial effusion.

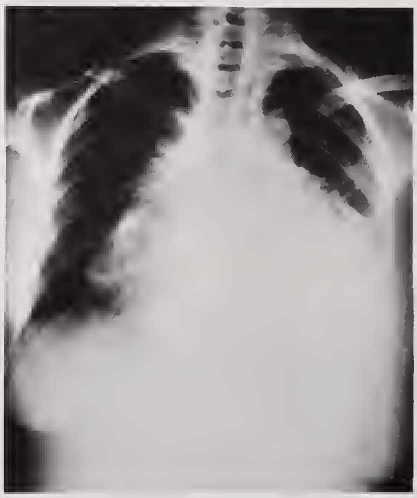


FIG. 24. Case 8. 2 years later the heart shadow is greatly enlarged. This was thought to be cardiomegaly, but the left hemidiaphragm is not seen through the heart shadow. Although the configuration of the heart is not entirely typical of pericardial effusion, an effusion was suspected because of obliteration of the diaphragm.

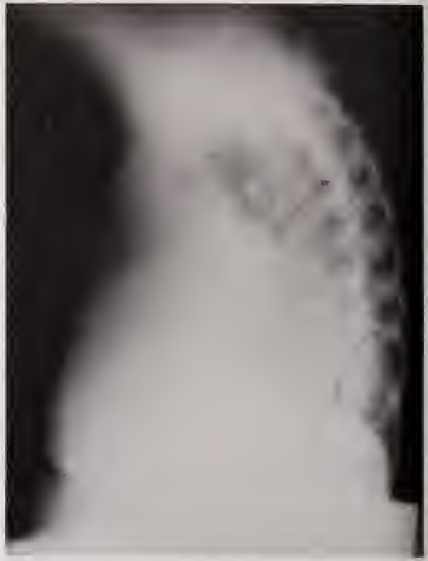


FIG. 25. Case 8. Upright lateral projection of chest shows no pulmonary disease or fluid to account for obliteration of diaphragm seen on upright frontal projection. This supported a diagnosis of pericardial effusion.

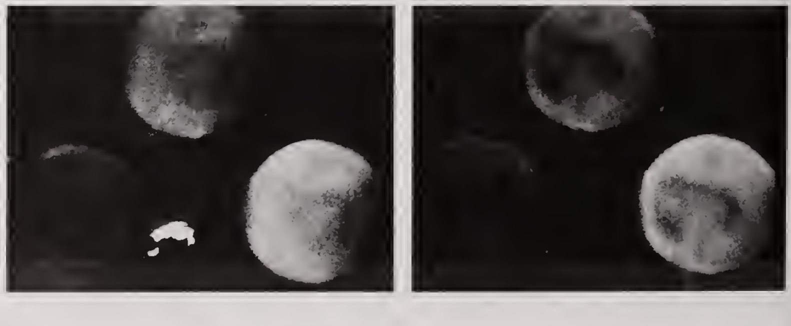


FIG. 26. Case 8. Heart-pool scan in frontal and lateral projections demonstrates a large pericardial effusion.

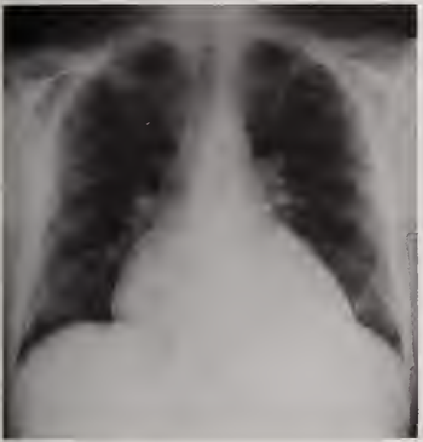


FIG. 27. Case 9. Cardiomegaly in a 43-year-old man with uremia and cirrhosis, proven at necropsy. Left hemidiaphragm is clearly outlined and seen through heart shadow.

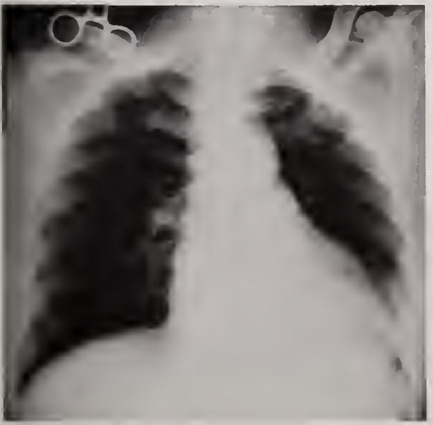


FIG. 28. Case 10. Cardiomegaly with left ventricular enlargement in a 46-year-old woman with old rheumatic valvculitis, proven at necropsy. Left hemidiaphragm is clearly outlined and seen through heart shadow.

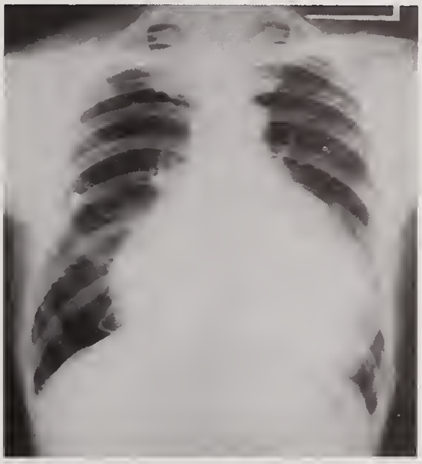


FIG. 29. Case 11. In 73-year-old man with a large, rather typical-appearing pericardial effusion in upright PA projection. Left hemidiaphragm has been obliterated and is not seen through heart shadow.

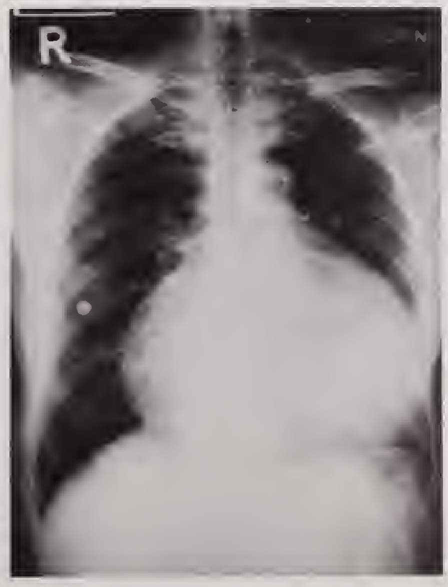


Fig. 30. Case 11. Configuration of pericardial effusion changes very little in supine projection, but left hemidiaphragm is now clearly seen through heart shadow. Change in left hemidiaphragm as a sign of pericardial effusion is thought to be reliable only in upright projection.

Summary and Conclusions

A helpful sign in the diagnosis of pericardial effusion is obliteration of the left hemidiaphragm, but a major restriction on this change is its dependence on a lateral chest film that demonstrates the left lower lobe to be free of pulmonary disease. This change has been described in the upright P-A projection, and alteration of the diaphragm as a plain-film sign of pericardial effusion is thought to be reliable only in this projection (Figs. 28 and 29). An underexposed chest radiogram may simulate a diminished or obliterated left hemidiaphragm, and this sign is dependent upon chest radiograms of good technical quality. In infants and small children a poor depth of inspiration will often efface the intracardiac diaphragm on plain chest X-ray films, presumably due to crowding of the pulmonary vessels and widening of the heart.

In practice, these limitations have not proven overly restrictive, and a number of enlarged hearts have been distinguished from pericardial effusions.

This sign is most helpful when combined with other plain-film signs of pericardial effusion, especially rapid increase in heart size with concomitant diminution of the left hemidiaphragm.

In the upright, P-A projection, within these limitations of this change in the left hemidiaphragm the following points are to be remembered:

1. If the diaphragm is clearly outlined within an enlarged cardiac silhouette, this is associated with cardiomegaly.
2. If the diaphragm is obliterated within an enlarged cardiac silhouette, a pericardial effusion should be excluded, even though a small number of these cases may be large hearts with dilated left ventricles.
3. If the diaphragm is diminished or effaced but not obliterated within an enlarged heart shadow, this may be

caused by the accumulation of pericardial fluid as well as an expanding left ventricle, and other clinical and radiographic signs of pericardial effusion should be sought.

Finally, it is hoped that this sign may help in selecting patients more discriminately for special procedures, especially the very young and the very ill. This is considered a preliminary report and depicts our experience with this change in the diaphragm to date.

Acknowledgement

Special thanks for continued guidance and assistance to Dr. Homer L. Twigg, Chairman, Department of Radiology, Georgetown University Hospital.

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On Prescribing Tranquilizers

LOUIS F. RITTELMEYER, JR., M.D.*

Tranquilizers are indicated for the control of anxiety, psychomotor hyperactivity and related or derivative symptoms. The special properties of these drugs do not lie in their ability to induce a unique psychic state different from sedation but in the dosage margins between their sedative effect and their effect on consciousness and physiologic functions. Classification of these agents is a simple one: there are major and minor tranquilizers.

The major group, consisting of the phenothiazine and rauwolfia derivatives, are characterized by: the production of emotional calmness with relatively little sedation, even in disturbed psychotic patients; reversible extrapyramidal symptoms and other toxic reactions, and little or no dependency or habituation.

The minor tranquilizers, of which meprobamate and chlordiazepoxide (Librium) are representative, do not produce a calming effect of the same quality as that of the major tranquilizers and are relatively ineffective in psychotic disorders. They have a low incidence of side effects but may be addictive. Barbiturates and alcohol also possess some properties of the minor group.

Major Tranquilizers

The major tranquilizers are capable of reducing extreme tension or agitation and may have an antipsychotic activity. They are primarily indicated for use in psychotic patients. The most effective major tranquilizers are the phenothiazine derivatives.

There are three types of phenothiazines. One subgroup includes chlorpromazine (Thorazine). These agents are quite sedative in their effects and are useful in controlling psychotic agitation and hyperactivity. Extrapyramidal reactions are common but these are reversible with the use of such antiparkinsonism drugs as trihexyphenidyl (Artane). Serious toxic effects occur rarely and then usually as a result of long-term use of very high doses.

Another subgroup includes thioridazine (Mellaril). It may be the most sedative of all phenothiazines. It is less likely to cause extrapyramidal side effects than other phenothiazines. Its most notable difference, however, is its usefulness in patients with agitated depression. Studies have shown it to be more effective than so-called antidepressants in patients who exhibit agitation with their depressive symptoms.

The third subgroup is the least sedative of all phenothiazines. Trifluoperazine and perphenazine are in this group. Their chief disadvantage is the high incidence of extrapyramidal symptoms.

It is not necessary to know the names of all the phenothiazines. It is better to become familiar with one from each subgroup. Their clinical similarities are much greater than their differences. For this reason, any of these drugs can be given to any psychotic patient with the one exception already noted, the severely depressed patient.

Rauwolfia alkaloids are no longer recommended for use in psychiatric patients. Their tendency to produce severe depression and to activate peptic ulcers is not balanced by any clear advantages.

Minor Tranquilizers

The minor tranquilizers include two groups of drugs. One consists of derivatives of the muscle relaxant mephenesin. They have the ability to relieve muscle spasm. In contrast to the phenothiazines, they have no effect on the autonomic nervous system. Also, they are habituating, which makes them extremely dangerous when given for chronic conditions or to persons with dependent or unstable personalities. The most popular drug in this group is meprobamate.

The second group consists of chlordiazepoxide and its analogues. These drugs are more potent than meprobamate, and they appear to have greater beneficial effects in relieving anxiety due to situational stress or psychoneurotic problems.

To these two groups may be added a third, miscellaneous group consisting of diphenhydramine (Benadryl), hydroxyzine, the barbiturates and alcohol. They have pharmacologic actions similar to those of the other minor tranquilizers, except that diphenhydramine and hydroxyzine do not seem to be habituating.

Habituation to the minor tranquilizers is dangerous primarily because of the patient's continuing dependency on them and his need for progressively larger doses. Another serious problem is the effect of sudden withdrawal. Convulsions and other symptoms may occur after sudden cessation of the drug.

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The main indication for the minor tranquilizers is the alleviation of anxiety. There is no question about the need for drug therapy if the symptoms are disabling. Where the incapacity is slight, however, it may be difficult to determine when tranquilizers should be used. A certain degree of tension or emotional tone is necessary for normal activity. It should be at a level to permit alertness—but not so high as to produce pain or so low as to impair effective functioning. The proper use of tranquilizers requires an understanding of this principle.

The widespread use of tranquilizers attests to the large numbers of people who appear to be suffering from excessive anxiety. Many physicians who are faced with troubled and anxious patients may simply not know what else to do for them. It would be more therapeutic for the physician to suppress the urge to "do something" immediately while he tries to understand what is happening in the person who seeks his help. If he does this, for even a few visits, he may discover that the patient's anxiety has subsided without the use of anything except that best, but often unrecognized, calming agent—the interested physician himself.

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Prescribing Information—Composition: Each white, beveled, compressed tablet contains: Quinine sulfate, 260 mg., Aminophylline, 195 mg. **Indications:** For the prevention and treatment of nocturnal and recumbency leg muscle cramps, including those associated with arthritis, diabetes, varicose veins, thrombophlebitis, arteriosclerosis and static foot deformities. **Contraindications:** Quinamm is contraindicated in pregnancy because of its quinine content. **Precautions/Adverse Reactions:** Aminophylline may produce intestinal cramps in some instances, and quinine may produce symptoms of cinchonism, such as tinnitus, dizziness, and gastrointestinal disturbance. Discontinue use if ringing in the ears, deafness, skin rash, or visual disturbances occur. **Dosage:** One tablet upon retiring. Where necessary, dosage may be increased to one tablet following the evening meal and one tablet upon retiring. **Supplied:** Bottles of 100 and 500 tablets.

Merrell
MERRELL-NATIONAL LABORATORIES
Division of Richardson-Merrell Inc.
Cincinnati, Ohio 45215

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Trademark: Quinamm

Quinamm
(quinine sulfate 260 mg., aminophylline 195 mg.)

Specific therapy for night leg cramps.

The Doctor as an "Employee"—Some Advantages and Disadvantages

The question of whether a doctor is an "employee" of the hospital where he may spend all or part of his working time is very important to such doctor from a tax standpoint. For example, if a doctor is an "employee", he may receive substantial tax savings if the "employer" hospital grants him stock options (in the case of a hospital owned by a "for profit" corporation) or pays the premiums due on insurance policies or annuities purchased for the doctor.

The case of annuities provides a good example of the importance to a doctor of his employee status. If the hospital of which he is an employee purchases an annuity policy for the doctor, the premiums paid for such policy by the hospital, whether deducted from or in addition to the other compensation due the doctor, will not be included in the doctor's taxable income. If the doctor is not an employee, the amount of such premiums would be included in the doctor's taxable income even though never actually paid to the doctor.

The question of whether a doctor, or any other professional, is an "employee", as the law defines that term, is more complex than one might initially believe. Traditionally, whether a person is considered an employee is based on the degree of control exerted over him by his employer. Thus, if the employer has the right to control and direct the details and means by which an individual accomplishes tasks for such employer, then such an individual will be an employee. Typical examples of employees are factory workers, clerical help and the like. It is important to note that it is the right to control the work that is the key element, as opposed to the degree of control actually exercised by the employer. Thus, if a doctor were to hire a lab technician, specifying the technician's exact duties and maintaining the prerogative to oversee his work, such a technician would be the doctor's employee even though the doctor as a practical matter did not review the technician's work on a regular basis.

If an individual is not considered an employee, then he will be considered an "independent contractor". An independent contractor is one who is given a specific task and expected to accomplish it using the particular expertise he has developed; the means by which an independent contractor accomplishes his tasks are not directly controlled. Thus, building contractors, consultants, collection agencies and the like would traditionally be classified as independent contractors.

The problem, of course, is that if these traditional definitions were strictly adhered to, then a doctor could never be an employee of a layman, because a layman would not ordinarily have the expertise to enable him to direct the details of a doctor's work. Thus, a doctor, if employed by lay hospital administrators, could never take advantage of the opportunities for tax savings available to "employees".

The result has been that in the case of doctors the definitions set forth above have been loosely applied. When the question has arisen, courts have looked for the various factors that normally accompany an employer-employee relationship; if many of these factors are present, then an employee status will be found.

As related directly to the hospital-doctor relationship, the doctor would be an employee of the hospital if the hospital,

> (1) paid the doctor a salary instead of compensating him on the basis of a percentage of fees generated;
>
> (2) withheld from the doctor's salary for Federal income tax purposes;
>
> (3) provided all the equipment, space and supplies needed by the doctor for the performance of work;
>
> (4) hired and compensated the doctor's assistants directly instead of having the doctor do so;
>
> (5) set fees generated by the doctor and billed for them on hospital stationery; and
>
> (6) intended the doctor to be its employee, and evidenced this intent by giving the doctor all or most of the fringe benefits of other employees, and by referring to his compensation as "salary" and his role in the hospital as a "position". In this respect, the wording of any employment contract between the hospital and doctor, and of any correspondence between them, will be extremely important.

Moreover, the doctor himself can do much to establish himself as an employee of the hospital. By not maintaining an office separate from the hospital, by not using personal stationery or cards, by making no announcement of his availability or the opening of his office, and by listing himself with the AMA as a hospital-based physician, the doctor will help establish his status as that of an employee. The above factors, as well as those noted in the previous paragraph, although not an exhaustive recitation, have been relied upon by courts in determining that the hospital has a high degree of control over the activities of the doctor, so that the doctor is an "employee". It should be stressed that no one of the factors noted above will be determinative of whether a doctor is an employee. The greater the number of such factors that are present, the higher the likelihood that a doctor will be an "employee", as that term is defined by the law.

If the doctor has determined that it would be advantageous to him from a tax standpoint to be considered an employee of a hospital, he may, for his own protection, wish to enter into an employment agreement with the hospital. Aside from the importance of the wording of such an employment contract noted above, it should also be noted that there are other factors unrelated to tax planning of which the parties should be aware in negotiating an employment contract. Because an employer is liable for the negligence and intentional misconduct of an employee, the hospital may wish to insert in such an employment contract provisions exculpating it from such liability. The hospital may also wish to include in such an employment contract provisions which would prevent the doctor from working for individuals or institutions other than the hospital. Thus, non-tax considerations from either the hospitals' or the doctor's point of view may outweigh the desirability of classifying the doctor an employee. A doctor who spends a substantial part of his time working in a hospital should therefore seek legal counseling if employee status appears to be significant.

MICHAEL H. LEAHY, J.D.

Edited by Stefan F. Tucker J.D., of Arent, Fox, Kintner, Plotkin & Kahn, Washington, D.C.

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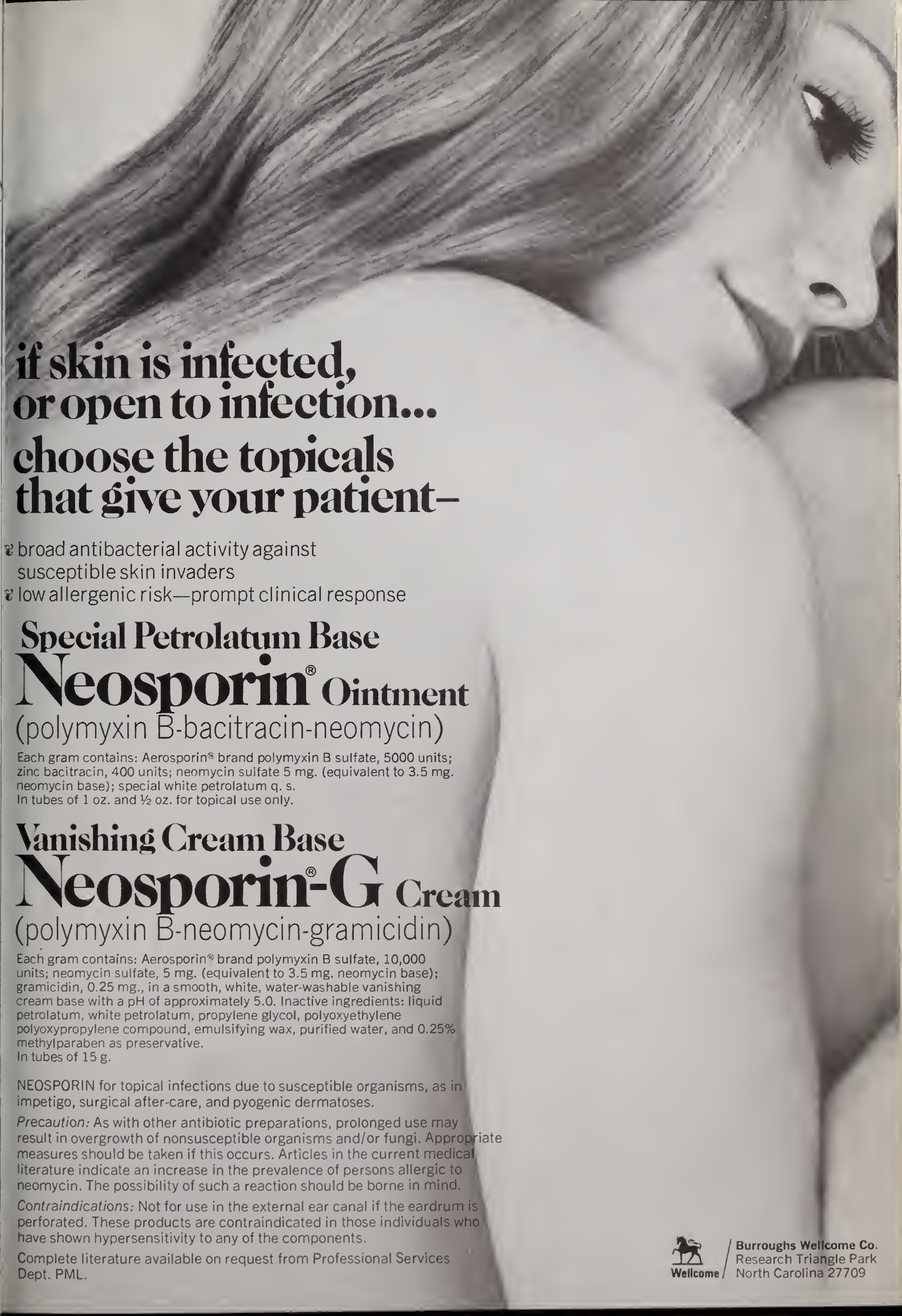

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choose the topicals that give your patient–

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Neosporin® Ointment

(polymyxin B-bacitracin-neomycin)

Each gram contains: Aerosporin® brand polymyxin B sulfate, 5000 units; zinc bacitracin, 400 units; neomycin sulfate 5 mg. (equivalent to 3.5 mg. neomycin base); special white petrolatum q. s.
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Each gram contains: Aerosporin® brand polymyxin B sulfate, 10,000 units; neomycin sulfate, 5 mg. (equivalent to 3.5 mg. neomycin base); gramicidin, 0.25 mg., in a smooth, white, water-washable vanishing cream base with a pH of approximately 5.0. Inactive ingredients: liquid petrolatum, white petrolatum, propylene glycol, polyoxyethylene polyoxypropylene compound, emulsifying wax, purified water, and 0.25% methylparaben as preservative.
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NEOSPORIN for topical infections due to susceptible organisms, as in impetigo, surgical after-care, and pyogenic dermatoses.

Precaution: As with other antibiotic preparations, prolonged use may result in overgrowth of nonsusceptible organisms and/or fungi. Appropriate measures should be taken if this occurs. Articles in the current medical literature indicate an increase in the prevalence of persons allergic to neomycin. The possibility of such a reaction should be borne in mind.

Contraindications: Not for use in the external ear canal if the eardrum is perforated. These products are contraindicated in those individuals who have shown hypersensitivity to any of the components.

Complete literature available on request from Professional Services Dept. PML.

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Beef Broth	25	Vegetable	77
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Chicken Gumbo	55	Cream of Chicken	87
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* From "Nutritive Composition of Campbell's Products" which gives values of important nutritive constituents of all Campbell's Products. For your copy, write to Campbell Soup Company, Dept. 536, Camden, New Jersey 08101.

There's a soup for almost every patient and diet ..for every meal and, it's made by Campbell

the 3 phases of Eve

All women are not equal in their endogenous hormonal output. And, while all oral contraceptives are fundamentally effective, they exhibit **differences in their activity levels and estrogen-progestogen ratios that affect different women differently**—in both short and long-term use. Some brands may be insufficient for the woman's needs or else may exceed them.

Searle offers a family of O.C. products that covers the range of women's needs to help you provide the right pill for the right woman at the right time.

the Ovulen phase

Most women* with a **balanced hormone profile an** **normal menses** do best on a middle-of-the-road pi that is neither estrogen dominant nor strongly progestogen dominant.

(*Typical clues—normal body build and breasts, feminine appearance, healthy skin and hair. Vagina cytology slide—balanced "pink and blue.")

Some women having problems on other O.C.s might do well on Ovulen.

Ovulen has a distinctive hormonal balance that combines moderate estrogenic activity with a sligh progestogen dominance. It has an excellent record of patient acceptance.

Ovulen®

Each white tablet contains: ethynodiol diacetate 1 mg./mestranol 0.1 mg.

References **1.** Editorial: Oral Contraceptives Which Pill for Which Patient? Patient Care *3* 90-115 (Feb.) 1969 and *4* 135-145 (June 15) 1970. **2.** Greenblatt, R. B. Progestational Agents in Clinical Practice, Med Sci *18* 37-49 (May) 1967. **3.** Kistner, R. W: Gynecology: Principles and Practice, ed 2, Chicago, Year Book Medical Publishers, 1971 **4.** Kistner, R. W The Pill. Facts and Fallacies About Today's Oral Contraceptives, New York, Delacorte Press, 1968 **5.** Nelson, J. H Clinical Evaluation of Side Effects of Current Oral Contraceptives, J. Reprod Med 6:50-55 (Feb.) 1971 **6.** Orr, G. W Oral Progestational Agents: Therapy and Complications, S Dakota J Med. *22* 11-17 (Jan.) 1969

SEARLE

For brief summary of prescribing information, see following page.

the Demulen phase

Many women* who secrete **more estrogen** than most do well on a pill with lower estrogen activity and an increased progestogen overbalance.

(*Typical clues—shorter, plumper, full-breasted, with glowing skin and no wrinkles. Vaginal cytology slide "pink.")

Some women with special conditions that may be aggravated by higher estrogen-activity products may do better on this ratio.

Demulen combines minimal estrogenic activity with a moderate ratio of progestogen overbalance. It is particularly well suited to the young when low-dose (activity) is preferred. Demulen offers little risk of the most potent progestogen side effects; early breakthrough bleeding is often transient.

Demulen®

Each white tablet contains ethynodiol diacetate 1 mg ethinyl estradiol 50 mcg

Each pink tablet in Ovulen-28® and Demulen® 28 is a placebo, containing no active ingredients.

Both Ovulen and Demulen are available in 21- and 28-pill schedules

the Enovid-E phase

Some women* who secrete **less estrogen** than most do best on a pill with a moderate estrogen overbalance.

(*Typical clues—oily complexion, acne, hirsutism, masculinity, flat chest. Vaginal cytology slide—"blue.")

Patients with estrogen deficiency may show:

premenopausal syndrome	intermittent depression
early-cycle bleeding	increased appetite
scanty menses	steady weight gain

vaginal candidiasis

Enovid-E not only provides increased estrogenic activity with low progestogen activity, but also contains the only progestogen that is not antiestrogenic. Therefore it offers less risk of high-dose progestogen side effects.

Enovid-E®

Each tablet contains norethynodrel 2.5 mg mestranol 0.1 mg

Oral contraceptives are complex medications and, after reference to the prescribing information, should be prescribed with discriminating care.

for the 3 phases of Eve:

a family of O.C. products

Ovulen®

Each white tablet contains.
ethynodiol diacetate 1 mg./mestranol 0.1 mg.

Demulen®

Each white tablet contains:
ethynodiol diacetate 1 mg./ethinyl estradiol 50 mcg.

Each pink tablet in Ovulen-28® and Demulen®-28 is a placebo, containing no active ingredients.

Actions—Ovulen and Demulen act to prevent ovulation by inhibiting the output of gonadotropins from the pituitary gland. Ovulen and Demulen depress the output of both the follicle-stimulating hormone (FSH) and the luteinizing hormone (LH).

Special note—Oral contraceptives have been marketed in the United States since 1960. Reported pregnancy rates vary from product to product. The effectiveness of the sequential products appears to be somewhat lower than that of the combination products. Both types provide almost completely effective contraception.

An increased risk of thromboembolic disease associated with the use of hormonal contraceptives has now been shown in studies conducted in both Great Britain and the United States. Other risks, such as those of elevated blood pressure, liver disease and reduced tolerance to carbohydrates, have not been quantitated with precision.

Long-term administration of both natural and synthetic estrogens in subprimate animal species in multiples of the human dose increases the frequency of some animal carcinomas. These data cannot be transposed directly to man. The possible carcinogenicity due to the estrogens can be neither affirmed nor refuted at this time. Close clinical surveillance of all women taking oral contraceptives must be continued.

Indication—Ovulen and Demulen are indicated for oral contraception.

Contraindications—Patients with thrombophlebitis, thromboembolic disorders, cerebral apoplexy or a past history of these conditions, markedly impaired liver function, known or suspected carcinoma of the breast, known or suspected estrogen-dependent neoplasia and undiagnosed abnormal genital bleeding.

Warnings—The physician should be alert to the earliest manifestations of thrombotic disorders (thrombophlebitis, cerebrovascular disorders, pulmonary embolism and retinal thrombosis). Should any of these occur or be suspected the drug should be discontinued immediately.

Retrospective studies of morbidity and mortality conducted in Great Britain and studies of morbidity in the United States have shown a statistically significant association between thrombophlebitis, pulmonary embolism, and cerebral thrombosis and embolism and the use of oral contraceptives. There have been three principal studies in Britain[1-3] leading to this conclusion, and one[4] in this country. The estimate of the relative risk of thromboembolism in the study by Vessey and Doll[3] was about sevenfold, while Sartwell and associates[4] in the United States found a relative risk of 4.4, meaning that the users are several times as likely to undergo thromboembolic disease without evident cause as nonusers. The American study also indicated that the risk did not persist after discontinuation of administration and that it was not enhanced by long-continued administration. The American study was not designed to evaluate a difference between products. However, the study suggested that there might be an increased risk of thromboembolic disease in users of sequential products. This risk cannot be quantitated, and further studies to confirm this finding are desirable.

Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions medication should be withdrawn.

Since the safety of Ovulen and Demulen in pregnancy has not been demonstrated, it is recommended that for any patient who has missed two consecutive periods pregnancy should be ruled out before continuing the contraceptive regimen. If the patient has not adhered to the prescribed schedule the possibility of pregnancy should be considered at the time of the first missed period.

A small fraction of the hormonal agents in oral contraceptives has been identified in the milk of mothers receiving these drugs. The long-range effect to the nursing infant cannot be determined at this time.

Precautions—The pretreatment and periodic physical examinations should include special reference to the breasts and pelvic organs, including a Papanicolaou smear since estrogens have been known to produce tumors, some of them malignant, in five species of subprimate animals. Endocrine and possibly liver function tests may be affected by treatment with Ovulen or Demulen. Therefore, if such tests are abnormal in a patient taking Ovulen or Demulen, it is recommended that they be repeated after the drug has been withdrawn for two months. Under the influence of progestogen-estrogen preparations preexisting uterine fibromyomas may increase in size. Because these agents may cause some degree of fluid retention, conditions which might be influenced by this factor, such as epilepsy, migraine, asthma, cardiac or renal dysfunction, require careful observation. In breakthrough bleeding, and in all cases of irregular bleeding per vaginam, nonfunctional causes should be borne in mind. In undiagnosed bleeding per vaginam adequate diagnostic measures are indicated. Patients with a history of psychic depression should be carefully observed and the drug discontinued if the depression recurs to a serious degree. Any possible influence of prolonged Ovulen or Demulen therapy on pituitary, ovarian, adrenal, hepatic or uterine function awaits further study. A decrease in glucose tolerance has been observed in a significant percentage of patients on oral contraceptives. The mechanism of this decrease is obscure. For this reason, diabetic patients should be carefully observed while receiving Ovulen or Demulen therapy. The age of the patient constitutes no absolute limiting factor, although treatment with Ovulen or Demulen may mask the onset of the climacteric. The pathologist should be advised of Ovulen or Demulen therapy when relevant specimens are submitted. Susceptible women may experience an increase in blood pressure following administration of contraceptive steroids.

Adverse reactions observed in patients receiving oral contraceptives—A statistically significant association has been demonstrated between use of oral contraceptives and the following serious adverse reactions: thrombophlebitis, pulmonary embolism and cerebral thrombosis.

Although available evidence is suggestive of an association, such a relationship has been neither confirmed nor refuted for the following serious adverse reactions: neuro-ocular lesions, e.g., retinal thrombosis and optic neuritis.

The following adverse reactions are known to occur in patients receiving oral contraceptives: nausea, vomiting, gastrointestinal symptoms (such as abdominal cramps and bloating), breakthrough bleeding, spotting, change in menstrual flow, amenorrhea during and after treatment, edema, chloasma or melasma, breast changes (tenderness, enlargement and secretion), change in weight (increase or decrease), changes in cervical erosion and cervical secretions, suppression of lactation when given immediately post partum, cholestatic jaundice, migraine, rash (allergic), rise in blood pressure in susceptible individuals and mental depression.

Although the following adverse reactions have been reported in users of oral contraceptives, an association has been neither confirmed nor refuted: anovulation post treatment, premenstrual-like syndrome, changes in libido, changes in appetite, cystitis-like syndrome, headache, nervousness, dizziness, fatigue, backache, hirsutism, loss of scalp hair, erythema multiforme, erythema nodosum, hemorrhagic eruption and itching.

The following laboratory results may be altered by the use of oral contraceptives: hepatic function: increased sulfobromophthalein retention and other tests; coagulation tests: increase in prothrombin, Factors VII, VIII, IX and X; thyroid function: increase in PBI and butanol extractable protein bound iodine, and decrease in T^3 uptake values; metyrapone test and pregnanediol determination.

References: 1. Royal College of General Practitioners: Oral Contraception and Thrombo-Embolic Disease, J. Coll. Gen. Pract. *13*:267-279 (May) 1967. **2.** Inman, W. H. W., and Vessey, M. P.: Investigation of Deaths from Pulmonary, Coronary, and Cerebral Thrombosis and Embolism in Women of Child-Bearing Age, Brit. Med. J. *2*:193-199 (April 27) 1968. **3.** Vessey, M. P., and Doll, R.: Investigation of Relation Between Use of Oral Contraceptives and Thromboembolic Disease. A Further Report, Brit. Med. J. *2*:651-657 (June 14) 1969. **4.** Sartwell, P. E.; Masi, A. T.; Arthes, F. G.; Greene, G. R., and Smith, H. E.: Thromboembolism and Oral Contraceptives: An Epidemiologic Case-Control Study, Amer. J. Epidem. *90*:365-380 (Nov.) 1969.

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Enovid-E®

norethynodrel 2.5 mg./mestranol 0.1 mg.

Actions—Enovid-E acts to prevent ovulation by inhibiting the output of gonadotropins from the pituitary gland. Enovid-E depresses the output of both the follicle-stimulating hormone (FSH) and the luteinizing hormone (LH).

Indication—Enovid-E is indicated for oral contraception.

The *Special Note, Contraindications, Warnings, Precautions* and *Adverse Reactions* listed above for Ovulen and Demulen are applicable to Enovid-E and should be observed when prescribing Enovid-E.

SEARLE **Enovid-E**®
brand of norethynodrel with mestranol

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Where "The Pill" Began

140/90 is normal blood pressure...or is it?

An extensive study based on nearly 4 million life insurance policies suggests that a blood pressure reading of 140/90 requires close medical supervision.

The Build
and
Blood Pressure
Study

Study Findings. Twelve years ago the Society of Actuaries reported on an extensive study based on the lives and deaths represented by almost 4 million life insurance policies. From this vast survey—"The Build and Blood Pressure Study"[1]—insurance experts concluded that:

• Blood pressure above 140/90 is accompanied by increased morbidity and requires close medical attention.

• Even small increments in either systolic or diastolic blood pressure progressively and steeply shorten life expectancy.

Other Studies. Studies conducted with large numbers of patients since that time have echoed the above findings. Two studies published in 1970—the VA Cooperative Study Group on "Effects of Treatment on Morbidity in Hypertension"[2] and the "Framingham Study"[3]—suggest that treatment of even mild hypertension may, over time, offer significant benefits to the patient.

Another Point of View. Although a growing body of studies suggests that treatment of mild hypertension is warranted, medical opinion is not unanimous. Some clinicians recommend that drug treatment for mild hypertension be reserved for patients with additional risk factors such as smoking, high cholesterol levels, heart or kidney involvement, or a family history of vascular disease. Dr. Walter M. Kirkendall stated this position in his recent paper "What's With Hypertension These Days?"[4] Discussing the management of hypertension in patients with a sustained diastolic pressure up to 100 mm Hg, he said: "Generally, I do not recommend antihypertensive therapy unless patient's blood pressure approaches the upper limit for the group and a number of adverse factors exist, such as male sex, family history of vascular disease, youth, evidence of heart or kidney involvement."

Drug Therapy for Hypertension. Although opinion varies on when to start drug therapy for mild hypertension, many physicians agree that treatment should start with a thiazide diuretic such as HydroDIURIL. For the adult patient, the usual starting dosage is 50 mg b.i.d. Dosage adjustments are recommended as the patient responds to treatment. The patient whose therapy begins with HydroDIURIL frequently can continue to benefit from it, because HydroDIURIL usually maintains its antihypertensive effect even when therapy is prolonged.

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25- and 50-mg tablets

HydroDIURIL®

(Hydrochlorothiazide|MSD)

Therapy to Start With

For a brief summary of prescribing information, please see next page.

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1. Society of Actuaries, *The Build and Blood Pressure Study*, 1959
2. Veterans Administration Cooperative Study Group on Antihypertensive Agents, "Effects of Treatment on Morbidity in Hypertension," JAMA *213* 1143-1152, Aug 17, 1970.
3. Kannel, William B., *et al* "Epidemiologic Assessment of the Role of Blood Pressure in Stroke—The Framingham Study," JAMA *214* 301-310, Oct. 12, 1970.
4. Kirkendall, Walter M.: "What's With Hypertension These Days?" Consultant, Jan 1971

25- and 50-mg tablets

HydroDIURIL®

(Hydrochlorothiazide|MSD)

Therapy to Start With

Drug Therapy for Hypertension. Although opinion varies on when to start drug therapy for mild hypertension, many physicians agree that treatment should start with a thiazide diuretic such as HydroDIURIL. For the adult patient, the usual starting dosage is 50 mg b.i.d. Dosage adjustments are recommended as the patient responds to treatment. The patient whose therapy begins with HydroDIURIL frequently can continue to benefit from it, because HydroDIURIL usually maintains its antihypertensive effect even when therapy is prolonged.

CONTRAINDICATIONS: Anuria; increasing azotemia and oliguria during treatment of severe progressive renal disease. Known sensitivity to this compound. Nursing mothers; if use of drug is deemed essential, patient should stop nursing.

WARNINGS: May precipitate or increase azotemia. Use special caution in impaired renal function to avoid cumulative or toxic effects. Minor alterations of fluid and electrolyte balance may precipitate coma in hepatic cirrhosis.

When used with other antihypertensive drugs, careful observation for changes in blood pressure must be made, especially during initial therapy. Dosage of other antihypertensive agents, especially ganglion blockers, must be reduced by at least 50% because HydroDIURIL potentiates their action.

Stenosis and ulceration of the small bowel causing obstruction, hemorrhage, and perforation have been reported with the use of enteric-coated potassium tablets, either alone or with nonenteric-coated thiazides. Surgery was frequently required, and deaths have occurred. Such formulations should be used only when indicated and when dietary supplementation is impractical. Discontinue immediately if abdominal pain, distention, nausea, vomiting, or gastrointestinal bleeding occurs.

Thiazides cross placenta and appear in cord blood. In women of childbearing age, potential benefits must be weighed against possible hazards to fetus, such as fetal or neonatal jaundice, thrombocytopenia, and possibly other adverse reactions which have occurred in the adult.

The possibility of sensitivity reactions should be considered in patients with a history of allergy or bronchial asthma. The possibility of exacerbation or activation of systemic lupus erythematosus has been reported for sulfonamide derivatives, including thiazides.

PRECAUTIONS: Check for signs of fluid and electrolyte imbalance, particularly if vomiting is excessive or patient is receiving parenteral fluids. Warning signs, irrespective of cause, are dryness of mouth, thirst, weakness, lethargy, drowsiness, restlessness, muscle pains or cramps, muscular fatigue, hypotension, oliguria, tachycardia, and gastrointestinal disturbances. Hypokalemia may develop (especially with brisk diuresis) in severe cirrhosis; with concomitant steroid or ACTH therapy; or with inadequate electrolyte intake. Digitalis therapy may exaggerate metabolic effects of hypokalemia, especially with reference to myocardial activity. Hypokalemia may be avoided or treated by use of potassium chloride or giving foods with a high potassium content. Similarly, any chloride deficit may be corrected by use of ammonium chloride (except in patients with hepatic disease) and largely prevented by a near normal salt intake. Hypochloremic alkalosis occurs infrequently and is rarely severe. In severely edematous patients with congestive failure or renal disease, a low salt syndrome may occur if dietary salt is unduly restricted, especially during hot weather.

Thiazides may increase responsiveness to tubocurarine. The antihypertensive effect of the drug may be enhanced in the postsympathectomy patient. Arterial responsiveness to norepinephrine is decreased, necessitating care in surgical patients. Discontinue drug 48 hours before elective surgery. Orthostatic hypotension may occur and may be potentiated by alcohol, barbiturates, or narcotics.

Pathological changes in the parathyroid glands with hypercalcemia and hypophosphatemia have been seen in a few patients on prolonged thiazide therapy. The effect of discontinuing thiazide therapy on serum calcium and phosphorus levels may be helpful in assessing the need for parathyroid surgery in such patients. Parathyroidectomy has elicited subjective clinical improvement in most patients, but has no effect on hypertension. Thiazide therapy may be resumed after surgery.

Use cautiously in hyperuricemic or gouty patients; gout may be precipitated. May affect insulin requirements in diabetics; may induce hyperglycemia and glycosuria in latent diabetics.

ADVERSE REACTIONS: Rare reactions include thrombocytopenia, leukopenia, agranulocytosis, aplastic anemia, cholestasis, and pericholangiolitic hepatitis. Nausea, vomiting, diarrhea, dizziness, vertigo, paresthesias, transient blurred vision, sialadenitis, purpura, rash, urticaria, photosensitivity, or other hypersensitivity reactions may occur. Cutaneous vasculitis precipitated by thiazide diuretics has been reported in elderly patients on repeated and continuing exposure to several drugs. Scattered reports have linked thiazides to pancreatitis, xanthopsia, neonatal thrombocytopenia, and neonatal jaundice. When adverse reactions are moderate or severe, the dosage of thiazides should be reduced or therapy withdrawn.

For more detailed information, consult your MSD Representative or see the Direction Circular. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486

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d.c. MENTAL HEALTH ADMINISTRATION

The Psychiatric Intensive Care Unit

Modern concepts of high quality psychiatric care are directed toward the goals of decreased institutional care, and the development of suitable alternatives to long-term hospitalization. It has been demonstrated repeatedly that brief hospitalization with early return of the patient to his community can be accomplished if high quality care is instituted with sufficient intensity to interrupt the progression of the illness before it reaches a chronic state. It is further recognized that the longer an individual is institutionalized, the less is the incidence of recovery.

The Mental Health Administration must be eternally alert to opportunities to incorporate new technologies and approaches to patient and client care. It is also necessary that we retain the latitude and flexibility needed to initiate new program designs in order that such innovations may be evaluated and assessed to determine their value in prevention, treatment, and rehabilitation of mental illness.

For these and other good and sufficient reasons, the Mental Health Administration is progressing toward the establishment of a specially designed operational unit whose mission will be directed toward the achievement of the following goals and objectives:

Crisis Intervention. It is frequently observed that acute psychiatric decompensation follows as a result of the inability of the individual's coping mechanism to adapt to crisis situations which may involve his social, vocational, economic, or interpersonal spheres. Under such circumstances there may be seen withdrawal from reality, bizarre behavior symbolically related to the activating stress, or a wide variety of aberrant behaviors which have been precipitated by the situation. Close attention and adequate therapy by a sympathethic staff can frequently abort this process, and allow rapid return of the individual to his community where referral can provide for continuing care through programs available in the appropriate CMHC. During this period of hospitalization, an in-depth study of his environment can be accomplished by social workers, psychiatric and psychometric evaluation can be performed, and a treatment plan individualized to the patient's needs.

Prevention of Hospitalization. Admission to the Psychiatric Intensive Care Unit would be made with the expectation that the duration of admission would not exceed three to five days. During this interval the patient would have access to a high staff/patient ratio which is of itself therapeutically beneficial. Opportunity would be available to the individual to describe and ventilate his problem, and with assistance to adapt sufficiently to avoid formalized hospitalization. The duration of hospitalization is often a reflection of administrative expectation, and this fact has been demonstrated repeatedly, if the maximum stay is a year, most patients stay a year; if it is two months, somehow every admission stays two months. If the expectation is that the duration is limited to a brief period, the results in time of discharge will be consistent. If referral to the twenty-four hour in-patient unit cannot be avoided, at least the stage will be set in the treatment plan so that the expectation will exist for shorter in-patient treatment. The entire climate will be geared to the expectation of rapid recovery and early release.

Collaborative Service to D.C. General Hospital. The authorities at the D.C. General Hospital have become painfully aware of the need for a service available to provide acute psychiatric care. The geographic proximity of this unit would increase our ability to serve this sister institution. Recent conferences between the officials of the hospital and representatives of the Mental Health Administration have served to emphasize the magnitude of this need.

In addition, the recently revised standards set forth by the Joint Commission on Accreditation of Hospitals requires the development of a written plan for the care or referral of persons needing mental health services.

The language used is as follows:

> "There should be a written plan for the care and/or for the referral of patients who are emotionally ill, or who become emotionally ill while in the hospital, as well as for the care and/or appropriate referral of persons who suffer the results of alcoholism or drug abuse. Medical staff responsibilities in this area must be clearly outlined."

The revised standards also state that "emergency patient care shall be guided by written policies, and shall be supported by appropriate procedural manuals and reference material". Such policies and procedures must include "instructions relative to the handling of persons who are emotionally ill, under the influence of drugs or alcohol . . ." This unit would also be available for referral of those patients already on the open units of the respective catchment areas whose condition may regress to the extent that they are in need of intensive short-term care. It would further be available for patients who may develop an acute psychiatric episode in the D.C. General Hospital, and who may require short periods of intensive psychiatric care prior to being returned to the main hospital.

Costs Effectiveness Considerations. Brief hospitalization as described in this paper will tend to supplant longer hospital stays for many disturbed persons, thus increasing the utilization of day care activities and other partial hospitalization programs. Cost accounting studies have revealed that prolonged twenty-four hour psychiatric hospitalization is the most expensive of all treatment modes. Costs for in-patient care are continually rising, and studies show that unless someone is mentally ill or has a family member who is mentally ill, that person seldom elects to pay insurance premiums for psychiatric care. With these considerations in mind, it becomes apparent that a significant savings might be realized through this approach. In the present climate in which funds for mental health care are becoming increasingly limited, it is imperative that methods be developed to obtain maximum utilization of staff and other resources. Any effort which succeeds in diminishing the amount of twenty-four hour hospitalization will contribute materially to these projected savings. The recent budget cuts imposed upon the District of Columbia Government lends added impetus to our efforts toward maximum utilization of existing staff and resources. Still another potential benefit will be realized through the increased utilization of paraprofessional personnel within the unit so that direct supervision could make them more effective extensions of the disciplined professional.

Adjunctive Service to Mobile Mental Health Units and the Office of Suicide Prevention Emergency Mental Health Consultation Services. The Mental Health Administration is simultaneously developing mobile mental health units whose function will include home visits and crisis intervention in the community. These are planned to respond to the needs of the Suicide Prevention and Emergency Mental Health Consultation Service as well as to the Metropolitan Police Department and other community agencies in need of their services. A suitable in-patient facility must be available for further evaluation on a short-term basis of patients they contact who are in need of additional study and treatment beyond that which can be delivered on the scene. In a like manner, referrals from the Suicide Prevention and Emergency Mental Health Consultation Service who are in need of short-term evaluation and treatment may also be served through the Psychiatric Intensive Care Unit.

Staffing Pattern Implications. Earlier reference has been made to the desirability of a high staff/patient ratio on this particular unit. With our present staff limitations it will be necessary that contributions of staff derive from the catchment areas served. The use of space at the Area C CMHC to initiate this unit is considered an interim arrangement. At such time as the Area B and A In-Patient Units are transferred to their respective centers, further modifications may be anticipated.

Consultation Services. It is expected that continuing exposure to the type of rapid evaluation to be performed in the Psychiatric Intensive Care Unit will develop a considerable expertise on the part of the staff. This expertise should develop an additional proficiency in the provision of psychiatric consultations to D.C. General Hospital and other District agencies in need of these services. These needs have been clearly defined and emphasized by officials of D. C. General Hospital, and the Mental Health Administration has the responsibility to respond to their needs. The rapid and effective provision of these services should improve and enhance the professional relations between agencies.

Finally, there is considerable potential for clinical experience in the training of psychologists, resident psychiatrists, psychiatric nurses, psychiatric social workers, and paraprofessionals involved in the delivery of mental health services.

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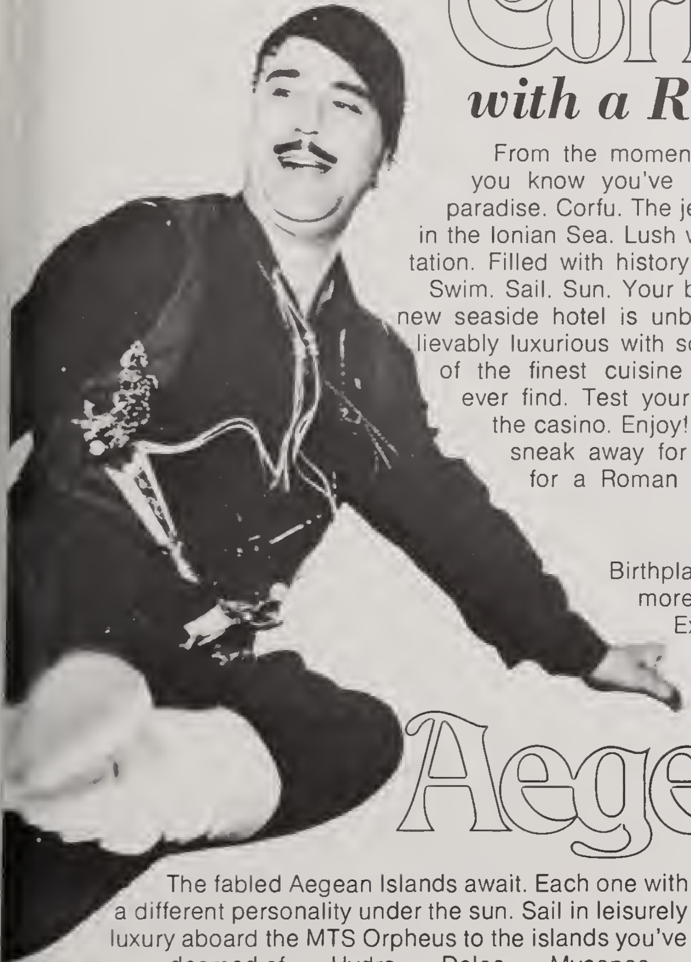

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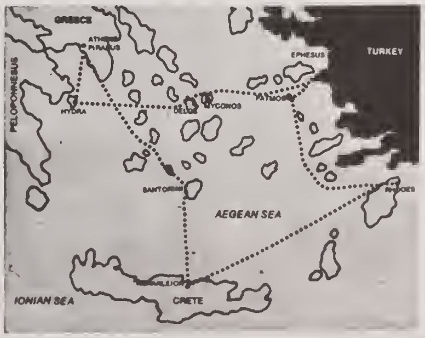

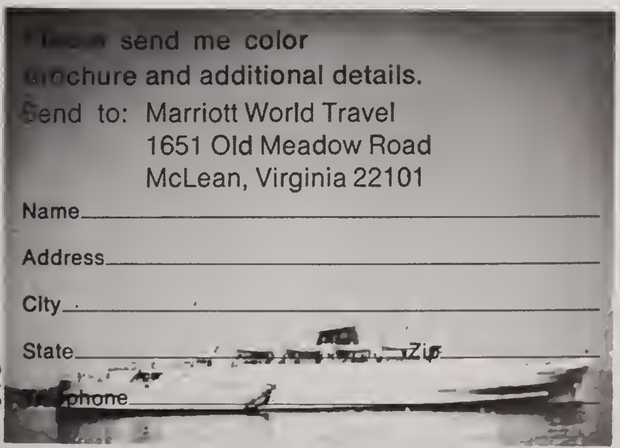

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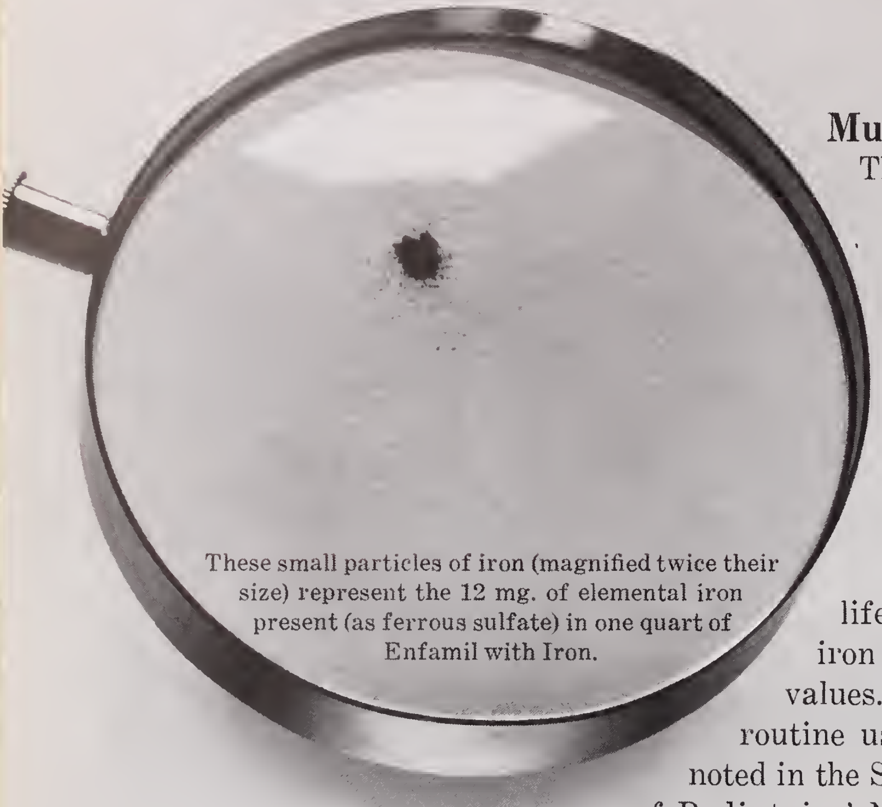

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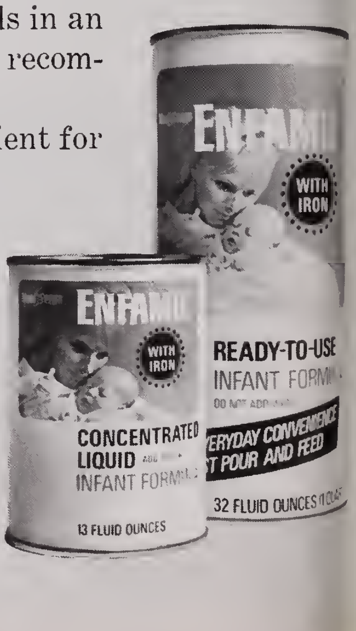

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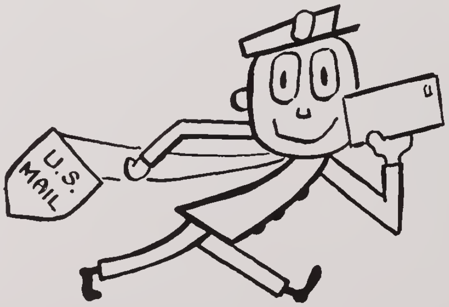

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book REVIEWS

High Altitude Physiology: Cardiac and Respiratory Aspects. RUTH PORTER AND JULIE KNIGHT, editors. Price, $7.20. Pp. 196. London: Churchill Livingstone, 1971.

This book is the result of a symposium on high altitude physiology held in 1971 in honor of Professor Alberto Hurtado of Peru for his pioneering and continuing work in physiology at high altitudes. Studies on the structure and function of the cardiorespiratory system, particularly work on the chemoreceptors and on myocardial metabolism at high altitude now reached the stage at which anatomists, physiologists and clinical investigators working in the subject could usefully meet to discuss their findings. Such were the circumstances under which this symposium was held. The latter further highlighted the importance of Dr. Hurtado's work in studying chronic adaptation or acclimatization as well as the acute effects of altitude.

The chapters on the pathophysiology of chronic mountain sickness and cor pulmonale were well written, though many questions remain unanswered. Unfortunately, although post mortem evidence on the effect of chronic mountain sickness would be desirable, such information is lacking. Doctors Severinghaus and West put forth some very fascinating concepts about periarterial cuffing and the site of origin of high-altitude pulmonary edema. Doctors Edwards' and Tenney's papers on chemoreceptors and their role in chronic adaptation certainly were enlightening and engendered much lively discussion.

What happens to a man at altitude is not only of interest to the high altitude physiologist but relevant to changes in patients with chronic lung disease and ischemic heart disease. The chapter on anatomy of the coronary circulation at high altitude by J. Arias-Stella and Marcello Topilsky is interesting. They showed that the number of branches leaving the main coronary trunks is greater and the peripheral ramifications are more numerous at high altitudes. Could these characteristics explain the infrequency of myocardial infarction in individuals from high altitude? More information of this nature together with the changes reported elsewhere in the monograph in coronary circulation, myocardial metabolism and biochemistry by P. R. Moret and Peter Harris could lead to a new understanding and treatment of these diseases in all humans.

This volume, though small, contains much valuable information that will be of interest to cardiologists, respiratory physiologists, biochemists as well as clinicians.

TSUNG O. CHENG, M.D.

Legal Medicine Annual: 1971. CYRIL H. WECHT, M.D., J.D., editor. Price: $14.50. Pp. 480. New York: Appleton-Century-Crofts, 1971.

This volume contains twenty-two contributions dealing with various aspects of forensic pathology, the physical forensic sciences and medical jurisprudence. The chapters are informative, and well written. In a field where there is no encyclopedic text, the Annual series, now in its third year, is a valuable reference source. There is something here for every practicing physician.

Due to the scope of the subject matter a few words regarding specific topics of current interest would seem appropriate. Medical review articles include the subjects of the sudden infant death syndrome ("crib death"), post-traumatic fat embolism, and the anatomic and circumstantial equivalents of death by homicidal smothering. The medical complications, clinical diagnosis and sequellae of cardiac contusion is an especially interesting chapter, dealing with an entity commonly overlooked or misdiagnosed by clinician and pathologist alike. Topics of more medical-legal relevance include various aspects of medical malpractice, standards of medical care, informed consent and transplantation.

The chapter entitled "Narcotic Abuse: A Medical Examiner's View" by Dr. Michael M. Baden presents an articulate, sensitive, and entirely accurate over-view of perhaps our number one public health dilemma. This contribution by itself is worth the price of the book. Given the facts that there are an estimated 20,000 heroin addicts in the District of Columbia and that narcotic-related fatalities are our second leading cause of death between the ages of 15 and 40 years, it is must reading.

JAMES L. LUKE, M.D.
Chief Medical Examiner
The District of Columbia

BOOK REVIEWS

What Shall We Tell the Kids? BENNETT OLSHAKER, M.D. Price, $6.95. Pp. 244. New York: Arbor House, 1971.

It is a curious phenomenon that after some seventy years of studying personality development and the factors underlying psychopathology, we still know relatively little about how to rear children. Much of what we do know is negative knowledge. That is, we know what causes trouble. We know that if you do such and such, there is a greater likelihood of children growing up with neurotic problems or bad behavior. Or, if you don't do something, if you leave out something important it can lead to emotional or intellectual difficulties.

Thus we can say that children need firmness, love, consistency, stimulation, example, and relationship, and that they should be protected from overstimulation, neglect, cruelty, separations, malnutrition, and excessive body manipulation. These, and a number of similar generalizations are the siftings out of the available data on child rearing.

Positive knowledge is the kind of information that would allow us to make predictions and plans. Someday, perhaps we will be able to say: if you want a child with such and such character traits or talents, then these are the practices you must engage in during his rearing; if you want a different pattern, then you must vary your tactics accordingly. Today, such a body of theory and technique is simply not available. At best, we know something, and a very limited something, about how to prevent trouble.

As meager as our information is, it is nonetheless true that a large segment of our population, including our professional population, doesn't even possess that. Or, if people do know it they keep that information out there, on an intellectual level, and don't really allow it to become meaningful, to affect what they actually do with their children.

For many people, the principles of child rearing that are advanced by mental health professionals have a distinctly peculiar ring. It doesn't seem "healthy minded" or "natural". If you talk to such people, they have a ready prescription: a lot of love and a few good whacks when he acts up, and a kid will be all right. Anecdotes follow to tell how they know a family where the parents raised 12 children and used the psychology book only to spank with, and they're all great people, etc.

Alas, they may indeed know such a family, but the chances are they don't know them well, for anyone who does know people well, knows that all too many of us stagger through our lives with very considerable burdens indeed. It is not a radical statement to assert that a large proportion of our population bears the stress of some diagnosable emotional or intellectual difficulty—indeed, it is only by accepting this statement that we can make any sense out of the commonly stated finding that one person in ten spends some time in psychiatric in-patient care in the course of his life.

In the light of such sobering figures, the matters alluded to in Dr. Olshaker's book, and the relatively meager body of knowledge available to us, assume new meaning. These are not merely desirable additions to our usual patterns of dealing with children—these become the critical distillations of decades of experience in treating troubled children, and they deserve the most careful consideration.

The content of Dr. Olshaker's book is by and large not new, and it is certainly not radical. It is extremely well written, a competent work, solid and communicative. In its attempt to be encyclopedic, many topics are treated briefly. But the sum of available information is not much greater than that included in these pages, and the essential data is here.

The topics covered range from sex, drugs, and divorce, to hospital care, the family, parental illness and the dying child. Dr. Olshaker thinks clearly, he is expert, and he informs well. He is not afraid to take a position and as such, he is open to certain areas of disagreement. But the performance is in general so sound, that it is less important to cavil than to applaud.

Where the difficulties lie becomes clear if we ask ourselves why works of this sort—there are others that have come before it and are widely read—seem to have so little impact on the culture in general. I think the answer lies in a peculiarity of human nature. People tend to learn about such highly emotional issues as how to raise one's child from other people. They learn from dialogues. Or from small group interactions. They learn through identification

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and through sharing feelings. And these are difficult experiences to go through while reading a book.

Here then is one of the chief uses of Dr. Olshaker's book: it would seem likely that in the long run it will achieve its greatest usefulness through informing the professional who will be having personal contacts with parents and who will be answering their questions. It is in short a good book for doctors.

JOSEPH D. NOSHPITZ, M.D.

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Of GENERAL INTEREST

DOCPAC

Each member of the District of Columbia Medical Society received an optional statement from DOCPAC with the annual dues statement. The initial response to this option has been disappointing. The importance of organized political activity in this calendar can scarcely be over-estimated. DOCPAC, the District of Columbia Political Action Committee, and its parent organization AMPAC, the American Medical PAC, represent the only voice of American medicine at the level of practical politics. Experienced politicians on Capitol Hill rate the political expertise of AMPAC equal to that of any other political action committee in the nation. Its continued effectiveness depends directly on the degree of support provided by its membership. If you have not already done so, I urge you to immediately send your membership check to DOCPAC at the D.C. Medical Society. $99 for a sustaining membership, $30 for a family membership, and $20 for an individual membership. Under legislation passed last year, political contributions are deductible items on your income tax return, up to a maximum of $100. Don't delay. Do it now! In November you'll be glad you did.

KARL C. JONAS, M.D.
Chairman
DOCPAC

obituaries

Claude Gilmore Walker, M.D.
1915–1972

Dr. Claude Gilmore Walker, a Washington area physician and former instructor at Howard University, died January 12, 1972. He resided at 1340 Girard Street, N.W., Washington, D.C.

A native of Frederick, Maryland, Dr. Walker received both his Bachelor of Science and Medical degrees from Howard University. He served his internship at Freedmen's Hospital, after which he entered the Army where he served as captain in the medical corps with the 93rd Infantry Division during the war years 1942–1946. He was awarded the Combat Medical Badge for action in the Solomon Islands.

Following World War II, Dr. Walker opened offices in the Community Medical-Dental Building at 3103 Georgia Avenue, N.W., where he engaged in the general practice of medicine. He taught anatomy at the Howard University Medical School from 1948 until 1957.

In addition to his membership in the Medical Society of the District of Columbia, Dr. Walker was a member of the National Medico-Chirurgical Society.

Dr. Walker is survived by his wife, Maude, of the home; a daughter, Nadine; two sons, Winston and Keith; his mother, Grace Washington, his father, Charles Gilmore Walker, and two sisters, Geraldine Reed and Clementine Johnson, all of Washington, D.C.

Eleanor E. Short, M.D.
1920-1972

Dr. Eleanor Elizabeth Short, a retired Washington area physician, died January 20, 1972. In private life she was Mrs. Robert Kneipp. She resided at 601 H St., N.W., Washington, D.C.

Born in Portage, Pennsylvania, Dr. Short was a graduate of Pennsylvania State University and received her M.D. degree from Temple University in Philadelphia. She interned at Western Pennsylvania Hospital in Pittsburgh, and served residencies at Norwich State Hospital in Connecticut, Grasslands Hospital in Valhalla, New York, and St. Elizabeth's Hospital in Washington, D.C.

Dr. Short held the position of Staff Psychiatrist at St. Elizabeth's Hospital 1949-1950, which she left in 1950 for a position with the District of Columbia Department of Public Health. She was Staff Psychiatrist with the Health Department's Bureau of Mental Hygiene 1950-1952, and held the same appointment in the Alcoholic Rehabilitation Division from 1952 to 1954. In 1954 she became Assistant Director of the Alcoholic Rehabilitation Division, which position she held at the time of her retirement in 1971.

In addition to her membership in the Medical Society of the District of Columbia, Dr. Short held membership in the American Psychiatric Association, the Washington Psychiatric Society, and the World Medical Association.

Dr. Short is survived by her mother, Mrs. Maude Hewitt Short of Portage, and a sister, Mrs. John Scoran of Ohio.

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> *"The history of science, and in particular the history of medicine . . . is . . . the history of man's reactions to the truth, the history of the gradual revelation of truth, the history of the gradual liberation of our minds from darkness and prejudice."*
>
> *—George Sarton, from "The History of Medicine Versus the History of Art"*

Should nongovernment scientists and physicians play a role in drug regulation?

Opinion

Results of a questionnaire to 7000 physicians:

82.8%
Physicians should play a role

78.3%
Independent scientists should play a role

69.8%
Medical academicians should play a role

Should nongovernment scientists and physicians play a role in drug regulation?

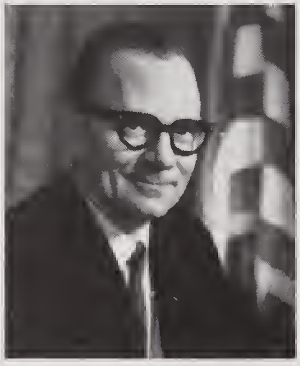
Doctor of Medicine

Herbert L. Ley, Jr.,
M.D., M.P.H., Formerly
Commissioner, F.D.A.
(1968-1969)
Currently Medical Consultant

In order for drug regulation to be effective, participation in the regulatory process from nongovernment physicians and scientists must be encouraged. Without such involvement, there will continue to be a high degree of controversy surrounding any regulations promulgated by the Food and Drug Administration.

There are two areas in which participation and communication by nongovernment physicians and scientists could significantly improve the process of regulation. First, scientists and physicians throughout the country could become involved in consulting relationships with the Food and Drug Administration in important scientific areas while regulatory policies are being evolved. If nongovernment professionals could bring their expertise and experience to bear early in the decision-making process, they would have less reason to criticize the final outcome.

Secondly, practicing physicians, academic physicians, and academic-based scientists could make it their business to comment on proposed regulations appearing in the Federal Register. Ideally, a system could be instituted whereby medical, scientific and technical people could see the Federal Register regularly, and provide the Food and Drug Administration with a body of opinion that has so far gone unheard. The FDA is caught among pressures from industry, Congress, the Presidential Administration and consumers. It should also feel pressures from practicing physicians and scientists.



In order to become more involved in these stages of the drug regulatory process, nongovernment physicians and scientists should begin to exercise their influence through their respective professional organizations, state and national medical societies, and specialty groups. Logically, a letter from these organizations representing a collective opinion has far greater weight in the regulatory process than individual letters. If the Food and Drug Administration receives opinions from these organizations early, before a regulation gets into the Federal Register, they are in a good position to respond with further study and review. Without such dissenting opinions, there is very little incentive to make changes in proposed regulations.

One instance in which practitioners did influence drug regulatory affairs in this way is the recent controversy that arose over the legitimacy of drug combinations. The strong opinion of practitioners on the value of such medication in clinical practice played a very prominent role in making the Food and Drug Administration modify its rather restrictive policy.

Another way in which practitioners can effectively influence drug regulations is by working with drug manufacturers conducting clinical trials of chemotherapeutic agents. When a drug is rated other than effective it may only mean that there is a lack of controlled clinical evidence as to efficacy. Thus, physicians might offer to conduct clinical studies that could help keep a truly effective drug in the marketplace. The treatment of diseases such as diabetes and angina are areas where the practitioner can aid in clinical studies because patients suffering from these diseases are rarely found in the conventional hospital setting.

By working with ethically and scientifically sound study designs in his everyday practice, the practitioner could begin to play an important part in determining official ratings on drug efficacy.

Nongovernment physicians and scientists and the FDA should also improve their lines of communication to the public. The medical community must develop a voice every bit as loud as that of the consumerists, the press, and others who sometimes criticize without complete information. If not, much of what the medical community *and* federal regulators do will often be represented in simplistic and somewhat misleading terms.

One illustration of the misuse of the media in this regard is the recall of anticoagulant drugs several years ago. This FDA action was given publicity by the press and television that went far beyond its probable importance. The result was a very uncomfortable situation for the practitioner who had patients taking these medications. Since the practitioner and pharmacist had not been informed of the action by the time it was publicized, in most states they were deluged with calls from worried patients.

The practitioner can attempt to solve these problems of inadequate communication in several ways. One would be the creation of a communications line in state pharmacy societies. When drug regulation news is to be announced, the society could immediately distribute a message to every pharmacist in the state. The pharmacist, in turn, could notify the physicians in his local community so that he and the physician could be prepared to answer inquiries from patients. Another approach would be to use professional publications the practitioner receives.

All of this leads back to my opening contention: if drug regulation is to be effective, timely, and related to the realities of clinical practice, a better method of communication and feedback must be developed between the nongovernmental medical and scientific communities and the regulatory agency.

One of a series

ue

Maker of Medicine

Henry W. Gadsden,
Chairman & Chief Executive Officer, Merck & Co., Inc.

In my opinion, it is the responsibility of all physicians and medical scientists to take whatever steps they think are desirable in a law- and regulation-making process that can have far-reaching impact on the practice of medicine. Yet many events in the recent past indicate that this is not happening. For example, it is apparent from drug efficacy studies that the NAS/NRC panels gave little consideration to the evidence that could have been provided by practicing physicians.

There are several current developments that should increase the concern of practicing physicians about drug regulatory affairs. One is the proliferation of malpractice claims and litigation. Another is the effort by government to establish the relative efficacy of drugs. This implies that if a physician prescribes a drug other than the "established" drug of choice, he may be accused of practicing something less than first-class medicine. It would come perilously close to federal direction of how medicine should be practiced.

In order to minimize this kind of arbitrary federal action, a way must be found to give practitioners both voice and representation in government affairs. Government must be caused to recognize the essentiality of seeking their views. One of the difficulties today, however, is that there is no way for concerned practitioners to participate in the early stages of decision-making processes. They usually don't hear about regulations until a proposal appears in the Federal Register, if then. By that time a lot of concrete has been poured, and a lot of boots are in the concrete.

Physicians in private practice, and particularly clinicians, should press for representation on the advisory committees of the Food and Drug Administration, joining with academic and teaching hospital physicians and scientists who are already serving. Though practitioners may not have access to all available information, the value of their clinical experience should be recognized. Clinicians, for example, rightly remind us that difficulty in *proving* precise effects does not necessarily mean a drug is ineffective.

Unless practitioners are more involved in drug regulations, it will be increasingly difficult for the pharmaceutical industry and scientists elsewhere to make optimal progress in drug development. The benefit/risk ratio must be re-emphasized, and as part of this it must be acknowledged that benefit can come from the judgments of medical science as a whole. Even this concept, unfortunately, is not always accepted in drug regulatory processes. For example, if current medical opinion holds that an excess of total lipids and cholesterol in the blood is probably predisposing to atherosclerosis, and if a drug is discovered which reduces total lipids and cholesterol, the drug ought to be accepted *prima facie* as a contribution to medical science . . . until someone disproves the theory. The sponsor should not have to prove the theory as well as to develop and test the drug.

I feel a major new effort must also be made to erase the feeling of mistrust of medicine and of medicines that seems to be growing in the public consciousness. Triggered primarily by strident announcements in Washington, people are reading and hearing confidence-shaking things almost continuously. Although challenge and awareness are essential to medical advancement, our long-term goal is constructively to build, not destroy. This means strengthening patient-physician relationships based on mutual confidence and trust. And in matters of health policy, it means working toward participatory rather than adversary proceedings—where everyone with an interest and a capacity to contribute has an opportunity to be heard . . . and, if that opportunity is not spontaneously afforded him, he may seek it.

Opinion & Dialogue

What is your opinion, doctor?
We would welcome your comments.

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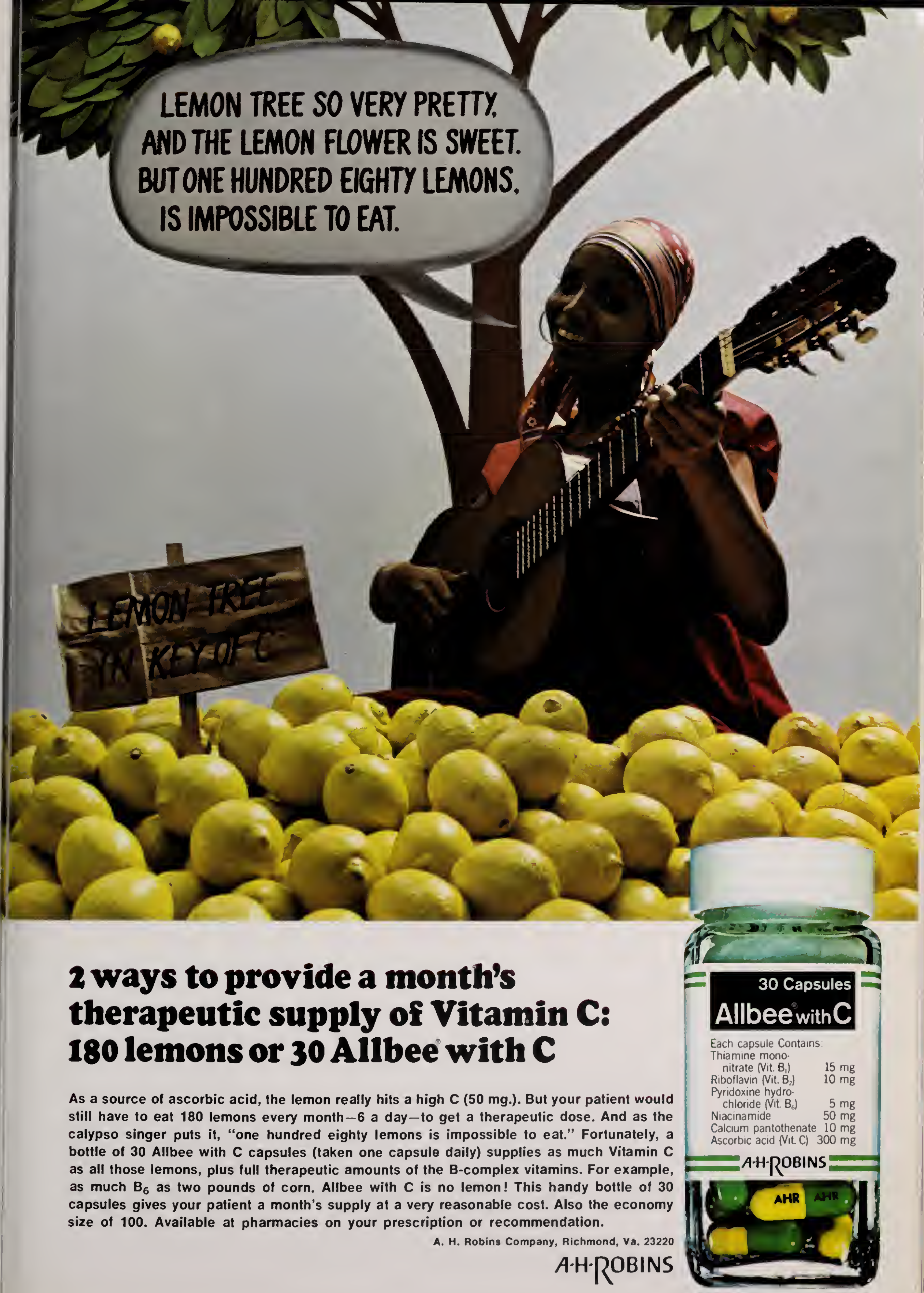
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(warning: may be habit forming)			

Brief summary. Side effects: Blurring of vision, dry mouth, difficult urination, and flushing or dryness of the skin may occur on higher dosage levels, rarely on usual dosage. Administer with caution to patients with incipient glaucoma or urinary bladder neck obstruction as in prostatic hypertrophy. Contraindicated in patients with acute glaucoma, advanced renal or hepatic disease or hypersensitivity to any of the ingredients.

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nportant Note: This drug is not a simple analgesic. o not administer casually. Carefully evaluate patients efore starting treatment and keep them under close upervision. Obtain a detailed history, and complete hysical and laboratory examination (complete emogram, urinalysis, etc.) before prescribing and at equent intervals thereafter. Carefully select patients, voiding those responsive to routine measures, con- aindicated patients or those who cannot be observed equently. Warn patients not to exceed recommended osage. Short-term relief of severe symptoms with e smallest possible dosage is the goal of therapy. osage should be taken with meals or a full glass of ilk. Patients should discontinue the drug and report mmediately any sign of: fever, sore throat, oral esions (symptoms of blood dyscrasia); dyspepsia, pigastric pain, symptoms of anemia, black or tarry tools or other evidence of intestinal ulceration or emorrhage, skin reactions, significant weight gain or dema. A one-week trial period is adequate. Discon- nue in the absence of a favorable response. Restrict reatment periods to one week in patients over sixty.
ndications: Acute gouty arthritis, rheumatoid arthritis, heumatoid spondylitis.
ontraindications: Children 14 years or less; senile atients; history or symptoms of G.I. inflammation or lceration including severe, recurrent or persistent yspepsia; history or presence of drug allergy; blood yscrasias; renal, hepatic or cardiac dysfunction; ypertension; thyroid disease; systemic edema; tomatitis and salivary gland enlargement due to the rug; polymyalgia rheumatica and temporal arteritis; atients receiving other potent chemotherapeutic gents, or long-term anticoagulant therapy.
arnings: Age, weight, dosage, duration of therapy, xistence of concomitant diseases, and concurrent otent chemotherapy affect incidence of toxic reac- ions. Carefully instruct and observe the individual atient, especially the aging (forty years and over) ho have increased susceptibility to the toxicity of the rug. Use lowest effective dosage. Weigh initially npredictable benefits against potential risk of severe, ven fatal, reactions. The disease condition itself is unaltered by the drug. Use with caution in first trimester of pregnancy and in nursing mothers. Drug may appear in cord blood and breast milk. Serious, even fatal, blood dyscrasias, including aplastic anemia, may occur suddenly despite regular hemograms, and may become manifest days or weeks after cessation of drug. Any significant change in total white count, relative decrease in granulocytes, appearance of immature forms, or fall in hematocrit should signal immediate cessation of therapy and complete hematologic investigation. Unexplained bleeding involving CNS, adrenals, and G.I. tract has occurred. The drug may potentiate action of insulin, sulfonylurea, and sulfonamide-type agents. Carefully observe patients taking these agents. Nontoxic and toxic goiters and myxedema have been reported (the drug reduces iodine uptake by the thyroid). Blurred vision can be a significant toxic symptom worthy of a complete ophthalmological examination. Swelling of ankles or face in patients under sixty may be prevented by reducing dosage. If edema occurs in patients over sixty, discontinue drug.
Precautions: The following should be accomplished at regular intervals: Careful detailed history for disease being treated and detection of earliest signs of adverse reactions; complete physical examination including check of patient's weight; complete weekly (especially for the aging) or an every two week blood check; pertinent laboratory studies. Caution patients about participating in activity requiring alertness and coordination, as driving a car, etc. Cases of leukemia have been reported in patients with a history of short- and long-term therapy. The majority of these patients were over forty. Remember that arthritic-type pains can be the presenting symptom of leukemia.
Adverse Reactions: This is a potent drug; its misuse can lead to serious results. Review detailed information before beginning therapy. Ulcerative esophagitis, acute and reactivated gastric and duodenal ulcer with perforation and hemorrhage, ulceration and perforation of large bowel, occult G.I. bleeding with anemia, gastritis, epigastric pain, hematemesis, dyspepsia, nausea, vomiting and diarrhea, abdominal distention, agranulocytosis, aplastic anemia, hemolytic anemia, anemia due to blood loss including occult G.I. bleeding, thrombocytopenia, pancytopenia, leukemia, leukopenia, bone marrow depression, sodium and chloride retention, water retention and edema, plasma dilution, respiratory alkalosis, metabolic acidosis, fatal and nonfatal hepatitis (cholestasis may or may not be prominent), petechiae, purpura without thrombocytopenia, toxic pruritus, erythema nodosum, erythema multiforme, Stevens-Johnson syndrome, Lyell's syndrome (toxic necrotizing epidermolysis), exfoliative dermatitis, serum sickness, hypersensitivity angiitis (polyarteritis), anaphylactic shock, urticaria, arthralgia, fever, rashes (all allergic reactions require prompt and permanent withdrawal of the drug), proteinuria, hematuria, oliguria, anuria, renal failure with azotemia, glomerulonephritis, acute tubular necrosis, nephrotic syndrome, bilateral renal cortical necrosis, renal stones, ureteral obstruction with uric acid crystals due to uricosuric action of drug, impaired renal function, cardiac decompensation, hypertension, pericarditis, diffuse interstitial myocarditis with muscle necrosis, perivascular granulomata, aggravation of temporal arteritis in patients with polymyalgia rheumatica, optic neuritis, blurred vision, retinal hemorrhage, toxic amblyopia, retinal detachment, hearing loss, hyperglycemia, thyroid hyperplasia, toxic goiter association of hyperthyroidism and hypothyroidism (causal relationship not established), agitation, confusional states, lethargy; CNS reactions associated with overdosage, including convulsions, euphoria, psychosis, depression, headaches, hallucinations, giddiness, vertigo, coma, hyperventilation, insomnia; ulcerative stomatitis, salivary gland enlargement.
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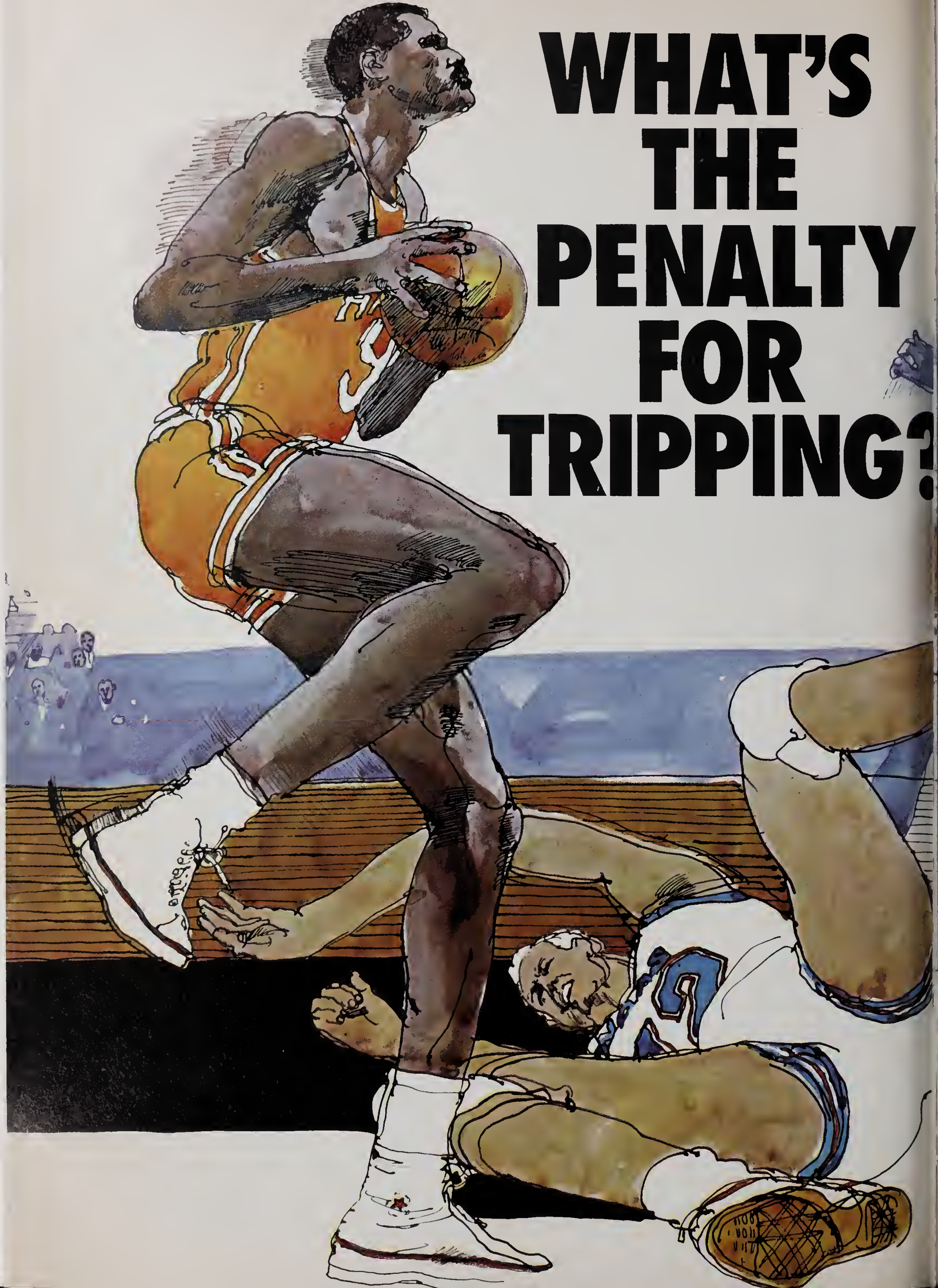

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Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence. In pregnancy, lactation or women of childbearing age, weigh potential benefit against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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Warnings: Do not use dietary potassium supplements or potassium salts unless hypokalemia develops or dietary potassium intake is markedly impaired. Enteric-coated potassium salts may cause small bowel stenosis with or without ulceration. Hyperkalemia (>5.4 mEq/L) has been reported in 4% of patients under 60 years, in 12% of patients over 60 years, and in less than 8% of patients overall. Rarely, cases have been associated with cardiac irregularities. Accordingly, **check serum potassium during therapy, particularly in patients with suspected or confirmed renal insufficiency (e.g., certain elderly or diabetics). If hyperkalemia develops, substitute a thiazide alone. If spironolactone is used concomitantly with 'Dyazide', check serum potassium frequently**—they can both cause potassium retention and sometimes hyperkalemia. Two deaths have been reported in patients on such combined therapy (in one, recommended dosage was exceeded; in the other, serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage or other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving Dyrenium (triamterene, SK&F). Rarely, leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with the thiazides. Watch for signs of impending coma in acutely ill cirrhotics. Thiazides are reported to cross the placental barrier and appear in breast milk. This may result in fetal or neonatal hyperbilirubinemia, thrombocytopenia, altered carbohydrate metabolism and possibly other adverse reactions that have occurred in the adult. When used during pregnancy or in women who might bear children, weigh potential benefits against possible hazards to fetus.

Precautions: Do periodic serum electrolyte and BUN determinations. Do periodic hematologic studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in postsympathectomy patients. The following may occur: hyperuricemia and gout, reversible nitrogen retention, decreasing alkali reserve with possible metabolic acidosis, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), digitalis intoxication (in hypokalemia). Use cautiously in surgical patients. Concomitant use with antihypertensive agents may result in an additive hypotensive effect.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis; rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting (may indicate electrolyte imbalance), diarrhea, constipation, other gastrointestinal disturbances. Rarely, necrotizing vasculitis, paresthesias, icterus, pancreatitis, and xanthopsia have occurred with thiazides alone.

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DZ-106

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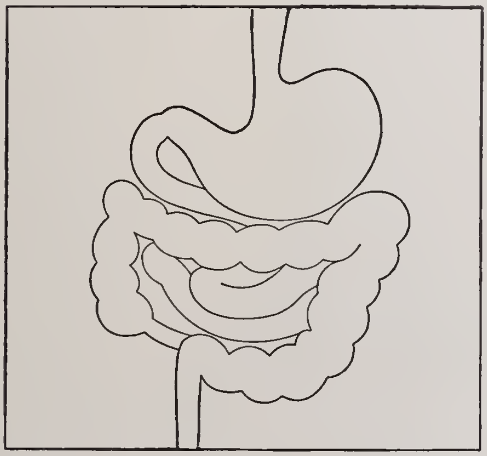

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can do more

Contraindications: Anticholinergics should not be used in patients with glaucoma, known prostatic hypertrophy, or pyloric obstruction. Urinary retention may indicate the presence of prostatic hypertrophy. If it occurs, the dose should be reduced or the drug withdrawn. Also contraindicated in patients with known hypersensitivity to one of the components.
Side Effects: Dryness of the mouth, blurred vision, dysuria, skin rash, constipation or drowsiness may occur.

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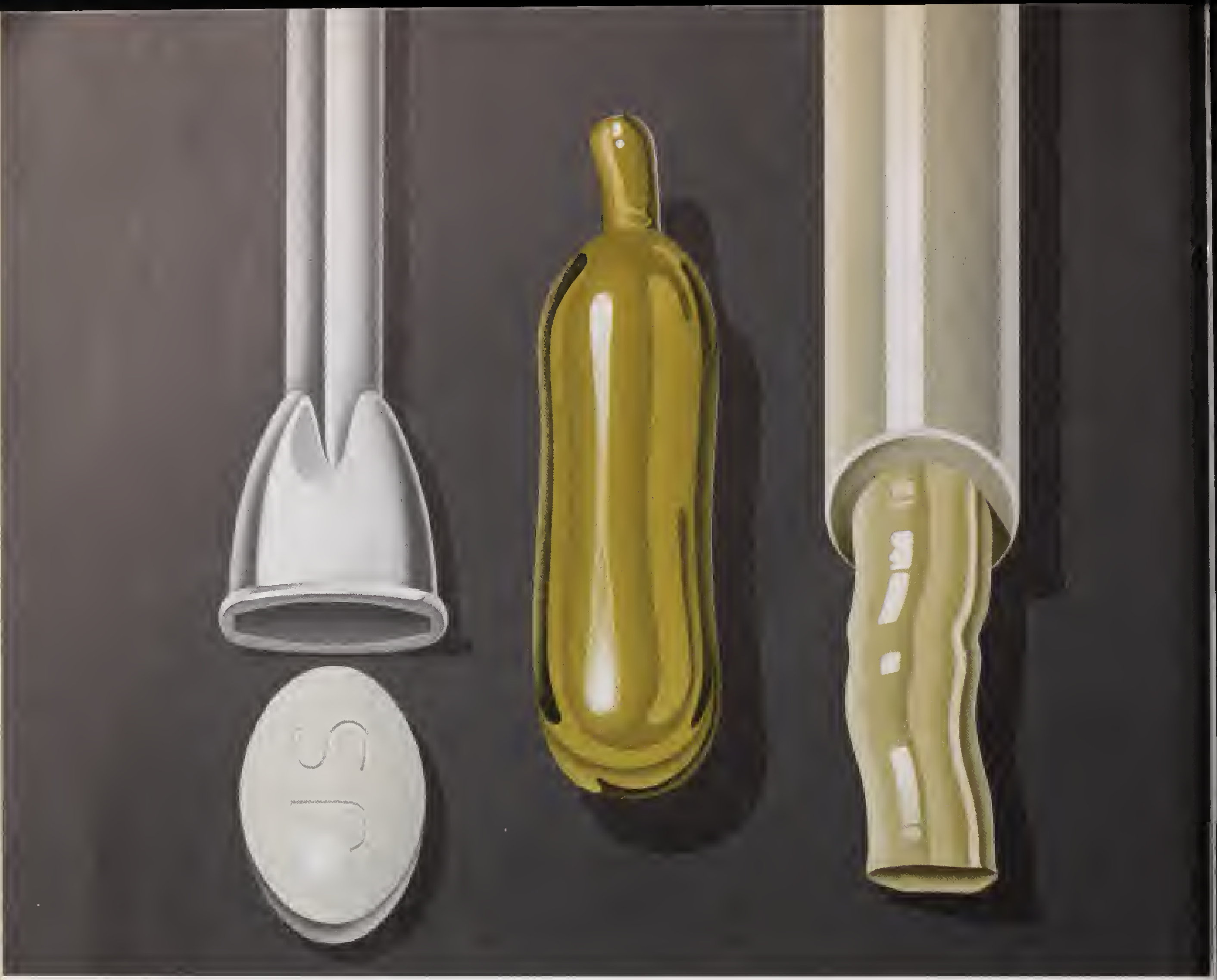

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CANDEPTIN®
(candicidin)

Description: CANDEPTIN (candicidin) Vaginal Ointment contains a dispersion of candicidin powder equivalent to 0.6 mg. per gm. or 0.06% Candicidin activity in U.S.P. petrolatum. 3 mg. of Candicidin is contained in 5 gm. of ointment or one applicatorful. CANDEPTIN Vaginal Tablets contain Candicidin powder equivalent to 3 mg. (0.3%) Candicidin activity dispersed in starch, lactose and magnesium stearate. CANDEPTIN VAGELETTES Vaginal Capsules contain 3 mg. of Candicidin activity dispersed in 5 gm. U.S.P. petrolatum.

Action: CANDEPTIN Vaginal Ointment, Vaginal Tablets, and VAGELETTES Vaginal Capsules possess anti-monilial activity.

Indications: Vaginitis due to Candida albicans and other Candida species.

Contraindications: Contraindicated for patients known to be sensitive to any of its components. During pregnancy manual Tablet or VAGELETTES Capsule insertion may be preferred since the use of the ointment applicator or tablet inserter may be contraindicated.

Caution: During treatment it is recommended that the patient refrain from sexual intercourse or the husband wear a condom to avoid re-infection.

Adverse Reaction: Clinical reports of sensitization or temporary irritation with CANDEPTIN Vaginal Ointment, Vaginal Tablets or VAGELETTES Vaginal Capsules have been extremely rare.

Dosage: One vaginal applicatorful of CANDEPTIN Ointment or one Vaginal Tablet or one VAGELETTES Vaginal Capsule is inserted high in the vagina twice a day, in the morning and at bedtime, for 14 days. Treatment may be repeated if symptoms persist or reappear.

Available Dosage Forms: CANDEPTIN Vaginal Ointment is supplied in 75 gm. tubes with applicator (14-day regimen requires 2 tubes). CANDEPTIN Vaginal Tablets are packaged in boxes of 28, in foil with inserter—enough for a full course of treatment. CANDEPTIN VAGELETTES Vaginal Capsules are packaged in boxes of 14 (14-day regimen requires 2 boxes.)

Store under refrigeration to insure full potency.

Federal law prohibits dispensing without prescription.

References: **1.** Olsen, J.R.: Journal-Lancet *85*: 287 (July) 1965. **2.** Giorlando, S.W.: Ob/Gyn Dig. *13*: 32 (Sept.) 1971. **3.** Decker, A.: Case Reports on File, Medical Department, Julius Schmid. **4.** Giorlando, S.W., Torres, J.F., and Muscillo, G.: Am. J. Obst. & Gynec. *90*: 370 (Oct. 1) 1964. **5.** Lechevalier, H.: Antibiotics Annual 1959-1960. New York, Antibiotica Inc., 1960. pp. 614-618. **6.** Friedel, H.J.: Maryland M.J., *15*:36 (Feb.) 1966.

Julius Schmid Pharmaceuticals
423 West 55th Street
New York, New York 10019

CANDEPTIN® (candicidin)

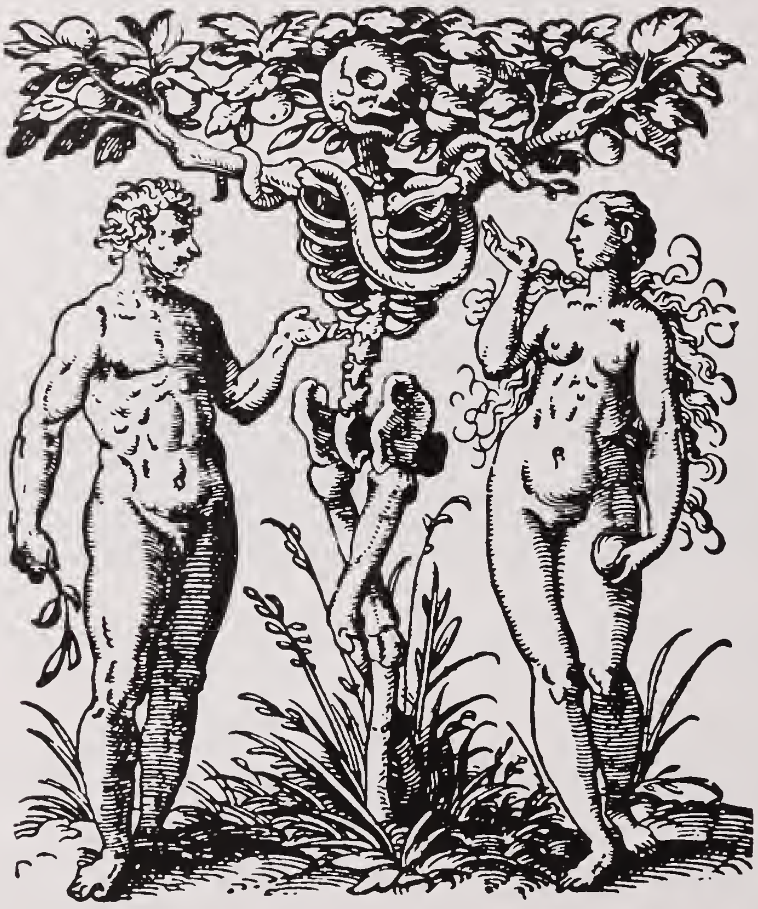
Vaginal Tablets

Vaginal Ointment

and VAGELETTES™ Vaginal Capsules

In acute gonorrhea

(urethritis, cervicitis, proctitis when due to susceptible strains of N. gonorrhoeae)



Sterile Trobicin®
(spectinomycin dihydrochloride pentahydrate)—For Intramuscular injections, 2 gm vials containing 5 ml when reconstituted with diluent. 4 gm vials containing 10 ml when reconstituted with diluent.

An aminocyclitol antibiotic active *in vitro* against most strains of *Neisseria gonorrhoeae* (MIC 7.5 to 20 mcg/ml). Definitive *in vitro* studies have shown no cross resistance of *N. gonorrhoeae* between Trobicin and penicillin.

Indications: Acute gonorrheal urethritis and proctitis in the male and acute gonorrheal cervicitis and proctitis in the female when due to susceptible strains of *N. gonorrhoeae*.

Contraindications: Contraindicated in patients previously found hypersensitive to Trobicin. Not indicated for the treatment of syphilis.

©1972 The Upjohn Company

Warnings: Antibiotics used to treat gonorrhea may mask delay the symptoms of incubating syphilis. Patients should carefully examined and monthly serological follow-up for least 3 months should be instituted if the diagnosis of syphilis suspected.

Safety for use in infants, children and pregnant women has n been established.

Precautions: The usual precautions should be observed wi atopic individuals. Clinical effectiveness should be monitored detect evidence of development of resistance of *N. gonorrhoe*

Adverse reactions: The following reactions were observ during the single-dose clinical trials: soreness at the injection si urticaria, dizziness, nausea, chills, fever and insomnia.

During multiple-dose subchronic tolerance studies in norm human volunteers, the following were noted: a decrease in hem

Trobicin®

sterile spectinomycin dihydrochloride pentahydrate, Upjohn

single-dose intramuscular treatment

---

igh cure rate:* 96% of 571 males, 95% of 294 females
osages, sites of infection, and criteria for diagnosis and cure are defined below.)**

ssurance of a single-dose, physician-controlled treatment schedule

o allergic reactions occurred in patients with an alleged history of penicillin sensitivity hen treated with Trobicin, although penicillin antibody studies were not performed

ctive against most strains of Neisseria gonorrhoeae in vitro (M.I.C. 7.5-20 mcg/ml)

single two-gram injection produces peak serum concentrations averaging about 0 mcg/ml in one hour (average serum concentrations of 15 mcg/ml present 8 hours after dosing)

ote: Antibiotics used in high doses for short periods of time to treat gonorrhea may mask or delay the mptoms of incubating syphilis. Since the treatment of syphilis demands prolonged therapy with any fective antibiotic, and since Trobicin is not indicated in the treatment of syphilis, patients being treated for onorrhea should be closely observed clinically. Monthly serological follow-up for at least 3 months should e instituted if the diagnosis of syphilis is suspected. Trobicin is contraindicated in patients previously found ypersensitive to it.

---

ata compiled from reports of 14 investigators. **Diagnosis was confirmed by cultural identification of *N. gonorrhoeae* on Thayer-ortin media in all patients. Criteria for cure: negative culture after at least 2 days post-treatment in males and at least 7 days post-eatment in females. Any positive culture obtained post-treatment was considered evidence of treatment failure even though the llow-up period might have been less than the periods cited above under "criteria for cure" except when the investigator determined at reinfection through additional sexual contacts was likely. Such cases were judged to be reinfections rather than relapses or ilures. These cases were regarded as non-evaluatable and were not included. JA72 1848-6

obin, hematocrit and creatinine clearance; elevation of alka-le phosphatase, BUN and SGPT. In single and multiple-dose udies in normal volunteers, a reduction in urine output was ted. Extensive renal function studies demonstrated no con-tent changes indicative of renal toxicity.

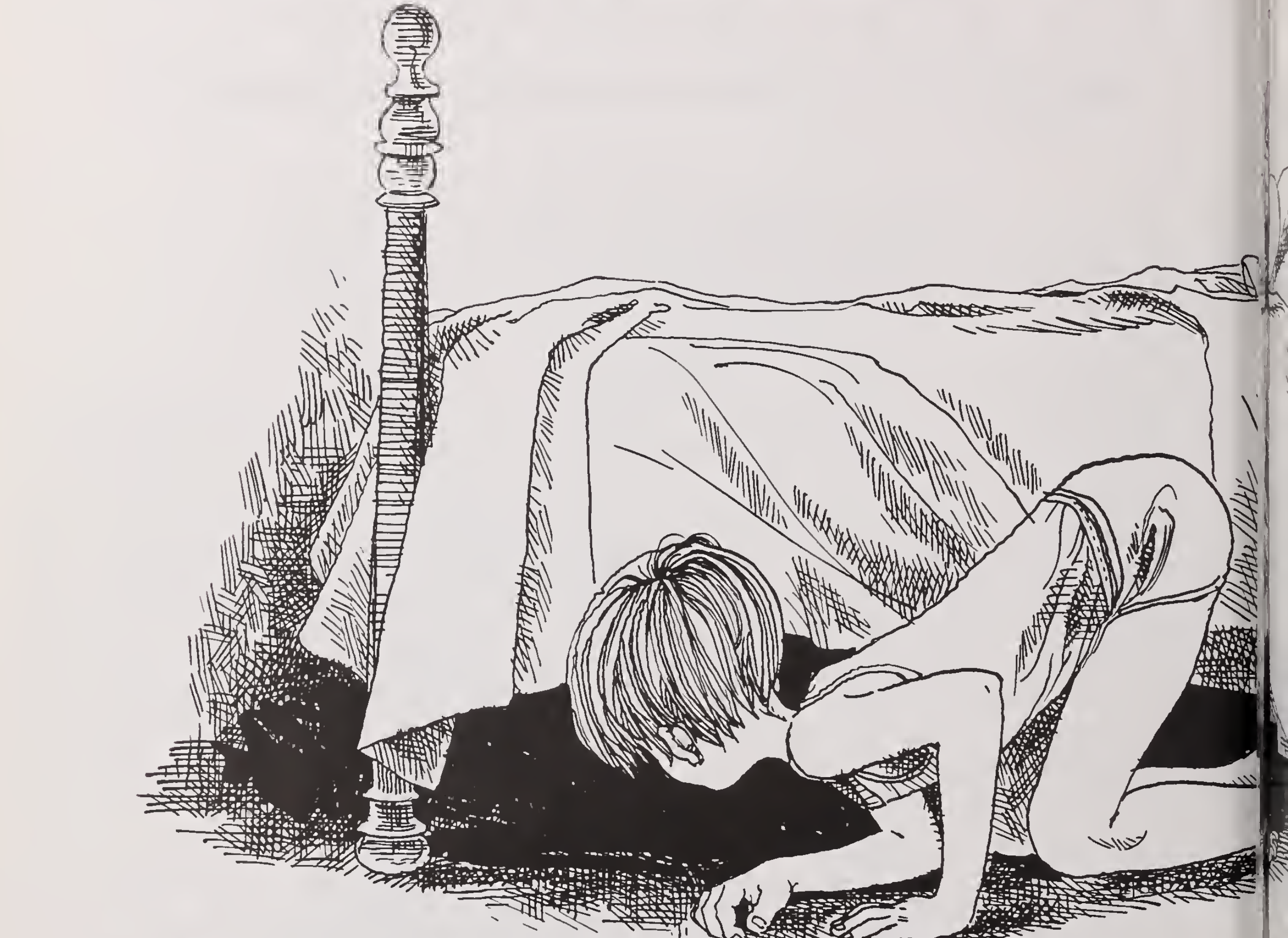
osage and administration: Keep at 25°C and use within hours after reconstitution with diluent.
ale—single 2 gram dose (5 ml) intramuscularly. Patients with onorrheal proctitis and patients being re-treated after failure previous antibiotic therapy should receive 4 grams (10 ml). In eographic areas where antibiotic resistance is known to be pre-lent, initial treatment with 4 grams (10 ml) intramuscularly is eferred.
male—single 4 gram dose (10 ml) intramuscularly.

ow supplied: *Vials, 2 and 4 grams—with ampoule of Bacteriostatic Water for Injection with Benzyl Alcohol 0.9% w/v.* Reconstitution yields 5 and 10 ml respectively with a concentration of spectinomycin dihydrochloride pentahydrate equivalent to 400 mg spectinomycin per ml. For intramuscular use only.
Susceptibility Powder—for testing *in vitro* susceptibility of *N. gonorrhoeae.*

Human pharmacology: Rapidly absorbed after intramuscular injection. A two-gram injection produces peak serum concentrations averaging about 100 mcg/ml at one hour with 15 mcg/ml at 8 hours. A four-gram injection produces peak serum concentrations averaging 160 mcg/ml at two hours with 31 mcg/ml at 8 hours.
For additional product information, see your Upjohn representative or consult the package insert. MED-B-1-S (LWB)

Upjohn The Upjohn Company, Kalamazoo, Michigan 49001

Arthur los



A single-dose, non-staining anthelmin

his pinworms...

with just one non-staining dose of Antiminth (pyrantel pamoate) Oral Suspension.

Highly effective. Active against pinworm...and roundworm.

Non-staining. Doesn't stain teeth or oral mucosa on ingestion. Doesn't stain stools, clothing or linen.

Simple dosage. Single-dose regimen: 1 cc. per 10 lbs. of body weight (1 tsp. per 50 lbs.).

Well-tolerated. Based on pre-introductory studies.

Pleasant-tasting. Easy-to-take, caramel-flavored oral suspension.

Economical. One prescription for the entire family.

Antiminth (pyrantel pamoate) Oral Suspension

Actions. Antiminth (pyrantel pamoate) has demonstrated anthelmintic activity against *Enterobius vermicularis* (pinworm) and *Ascaris lumbricoides* (roundworm). The anthelmintic action is probably due to the neuromuscular blocking property of the drug.

Antiminth is partially absorbed after an oral dose. Plasma levels of unchanged drug are low. Peak levels (0.05-0.13 $\mu g/ml$) are reached in 1-3 hours. Quantities greater than 50% of administered drug are excreted in feces as the unchanged form, whereas only 7% or less of the dose is found in urine as the unchanged form of the drug and its metabolites.

Indications. For the treatment of ascariasis (roundworm infection) and enterobiasis (pinworm infection).

Warnings. *Usage in Pregnancy:* Reproduction studies have been performed in animals and there was no evidence of propensity for harm to the fetus. The relevance to the human is not known.

There is no experience in pregnant women who have received this drug.

Precautions. Minor transient elevations of SGOT have occurred in a small percentage of patients. Therefore, this drug should be used with caution in patients with pre-existing liver dysfunction.

Adverse Reactions. The most frequently encountered adverse reactions are related to the gastrointestinal system.

Gastrointestinal and hepatic reactions: anorexia, nausea, vomiting, gastralgia, abdominal cramps, diarrhea and tenesmus, transient elevation of SGOT.

CNS reactions: headache, dizziness, drowsiness, and insomnia.

Skin reactions: rashes.

Dosage and Administration. *Children and Adults:* Antiminth Oral Suspension (50 mg. of pyrantel base/ml.) should be administered in a single dose of 11 mg. of pyrantel base per kg. of body weight (or 5 mg./lb.); maximum total dose 1 gram. This corresponds to a simplified dosage regimen of 1 cc. of Antiminth per 10 lbs. of body weight. (One teaspoonful = 5 cc.)

Antiminth (pyrantel pamoate) Oral Suspension may be administered without regard to ingestion of food or time of day; and purging is not necessary prior to, during, or after therapy. It may be taken with milk or fruit juices. Because of limited data on repeated doses, no recommendations can be made.

How Supplied. Antiminth is available as a pleasant tasting caramel-flavored suspension which contains the equivalent of 50 mg. pyrantel base per ml., supplied in 60 cc. bottles.

gent: **new ANTIMINTH®**
(pyrantel pamoate)
equivalent to 50 mg pyrantel/ml
ORAL SUSPENSION

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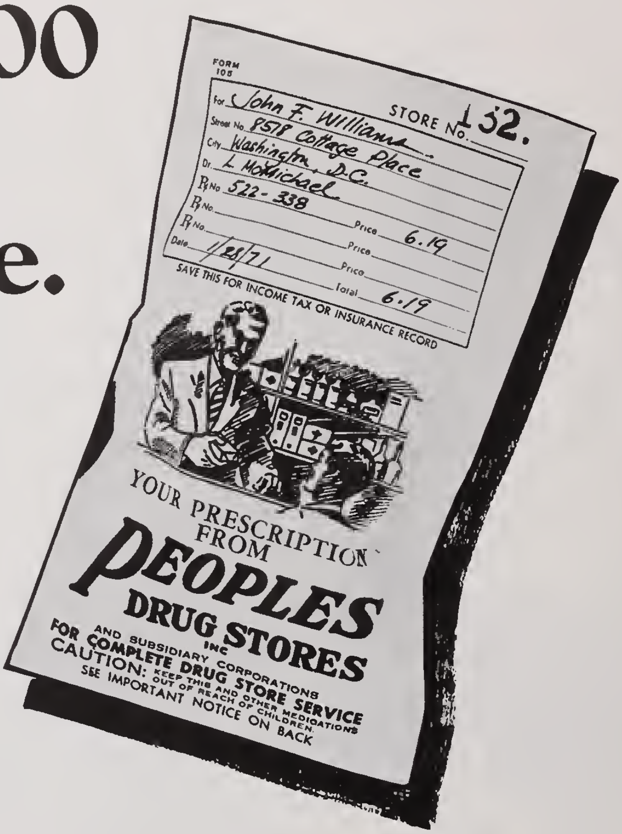
The bag that has earned more than 125,000,000 votes of confidence.



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Nothing new about Synirin other than …it's a stable, uncoated, fast disintegrating tablet of aspirin with pentobarbital potentiating the aspirin analgesia.

Synirin®

ASPIRIN 5 GR.—PENTOBARBITAL 1/8 GR.

ETHICAL ANALGESIA (economical if prescribed in 100 units with privilege of refills)

PRESCRIBING INFORMATION: To relieve tension headaches and arthritic pains, 2 tablets q 4 h. Aspirin and pentobarbital begin their action promptly, continuing for about 4 hours. The small pentobarbital content gives no perceptible sedation. Pentobarbital is destroyed by the body and there is no accumulation. Synirin will supply any aspirin therapy with equal safety. Use aspirin with caution in peptic ulcer.

EACH UNCOATED TABLET CONTAINS:

Aspirin ....325 mg. (5 gr.)
Pentobarbital* ....8 mg. (1/8 gr.)

*May be habit forming.

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DISPENSED IN BOTTLES OF 100 AND 1000 TABLETS

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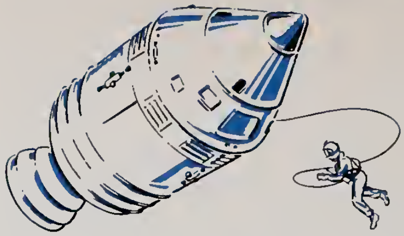
A clinical supply of this new aspirin formulation may be requested.



Man in space, now fait accompli, re-emphasizes the importance of Uro-Phosphate therapy. Research into the effect of space travel on the astronaut reveals that weightlessness causes loss of bone calcium. As the bones are required to bear less and less of the weight of the body they lose calcium, increasing the calcium content of the urine. When physical activity is reduced, the acidity of the urine should be adjusted to keep increased calcium in solution . . . . a prophylaxis to prevent kidney or bladder calculi.

Uro-Phosphate®

NOW A SUGAR-COATED TABLET

Each tablet contains: METHENAMINE, 300 mg.; SODIUM ACID PHOSPHATE, 500 mg.

Uro-Phosphate gives comfort and protection when inactivity causes discomfort in the urinary function. It keeps calcium in solution, preventing calculi; it maintains clear, acid, sterile urine; it encourages complete voiding and lessens frequency when residual urine is present.

Dosage:

For protection of the inactive patient 1 or 2 tablets every 4 to 6 hours is usually sufficient to keep the urine clear, acid and sterile.

2 tablets on retiring will keep residual urine acid and sterile, contributing to comfort and rest.

A clinical supply will be sent to physicians and hospitals on request.

Uro-Phosphate contains sodium acid phosphate, a natural urinary acidifier. This component is fortified with methenamine which is inert until it reaches the acid urinary bladder. In this environment it releases a mild antiseptic keeping the urine sterile.

Uro-Phosphate is safe for continuous use. There are no contra-indications other than acidosis. It can be given in sufficient amount to keep the urine clear, acid and sterile. A heavy sugar coating protects its potency.

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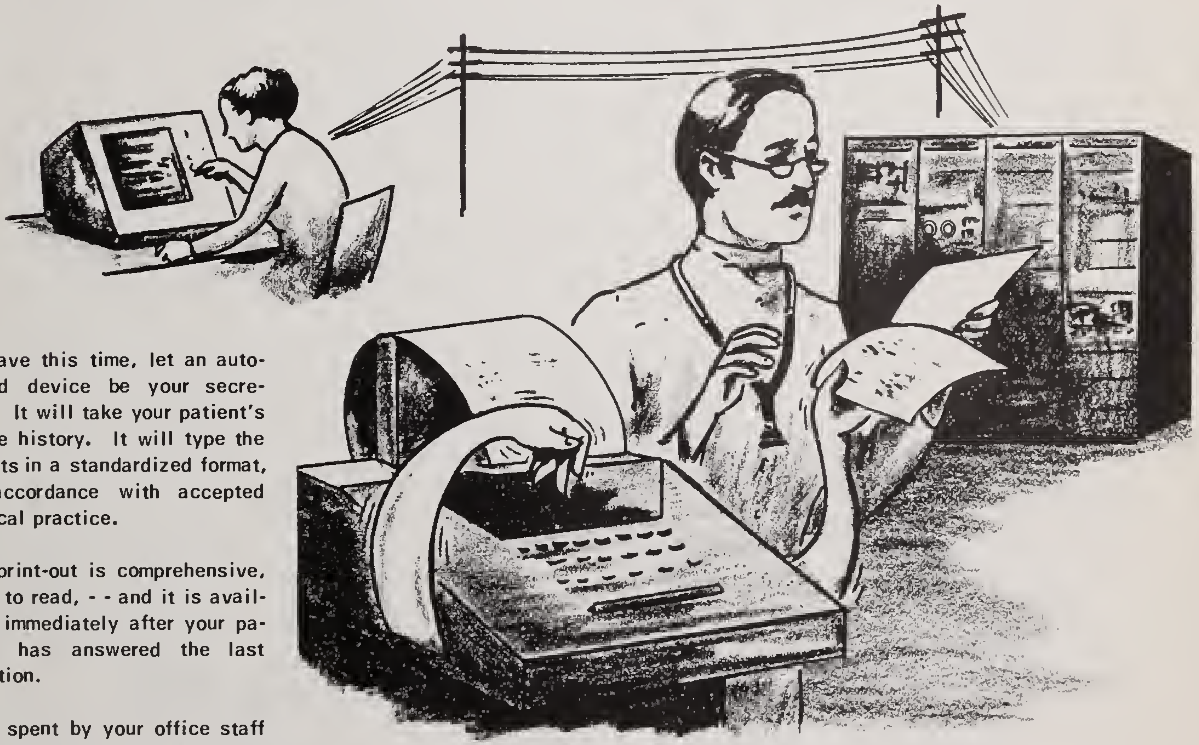

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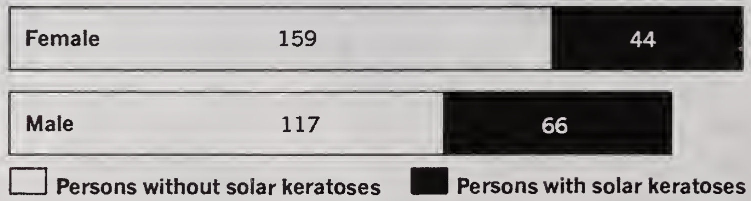
What it means to live and work in Tipton County, Tennessee

Persons who are white and over 40 have one chance in four of having solar keratoses... which may be premalignant

An epidemiologic study* conducted in Tipton County, Tennessee, revealed that 28.5% of white persons over 40 had solar keratoses; most had multiple lesions. Cluster sampling projected an estimated prevalence of 32.5% for white males and 19.5% for white females.

Though this is an unusually high percentage of affected persons, these lesions can occur in any white population, wherever people work or play out of doors.

Prevalence of solar keratoses in white persons over 40 in Tipton County, Tennessee



*Data on file, Hoffmann-La Roche Inc., Nutley, New Jersey.

Solar, actinic, senile keratoses

Called by many names, the typical lesion is flat or slightly elevated, brownish or reddish in olor, papular, dry, adherent, rough, sharply efined; usually multiple lesions, chiefly on xposed portions of the skin.

Sequence/selectivity of response

Erythema in areas of lesions may begin after everal days of therapy; height of reaction only in affected areas)* usually occurs within wo weeks, declining after discontinuation of herapy. Since this response is so predictable, esions that do not respond should be biopsied o rule out the presence of a frank neoplasm.

Cosmetic results

Cosmetic results are highly favorable. Incilence of scarring is low—important with multiple facial lesions. Efudex should be applied with care near the eyes, nose and mouth.

5% cream–a Roche exclusive

Only Roche formulates the 5% cream... high in patient acceptability...high in clinical efficacy, especially for lesions of hands and forearms...economical.

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Multiple actinic or solar keratoses.
Contraindications: Patients with known hypersensitivity to any of its components.
Warnings: If occlusive dressing used, may increase inflammatory reactions in adjacent normal skin. Avoid prolonged exposure to ultraviolet rays. Safe use in pregnancy not established.
Precautions: If applied with fingers, wash hands immediately. Apply with care near eyes, nose and mouth. Lesions failing to respond or recurring should be biopsied.
Adverse Reactions: Local—pain, pruritus, hyperpigmentation and burning at application site most frequent; also dermatitis, scarring, soreness and tenderness. Also reported—insomnia, stomatitis, suppuration, scaling, swelling, irritability, medicinal taste, photosensitivity, lacrimation, leukocytosis, thrombocytopenia, toxic granulation and eosinophilia.
Dosage and Administration: Apply sufficient quantity to cover lesion twice daily with nonmetal applicator or suitable glove. Usual duration of therapy is 2 to 4 weeks.
How Supplied: Solution, 10-ml drop dispensers—containing 2% or 5% fluorouracil on a weight/weight basis, compounded with propylene glycol, tris(hydroxymethyl)aminomethane, hydroxypropyl cellulose, parabens (methyl and propyl) and disodium edetate.
Cream, 25-Gm tubes—containing 5% fluorouracil in a vanishing cream base consisting of white petrolatum, stearyl alcohol, propylene glycol, polysorbate 60 and parabens (methyl and propyl).

an alternative to conventional therapy

Efudex®
(fluorouracil)
cream/solution

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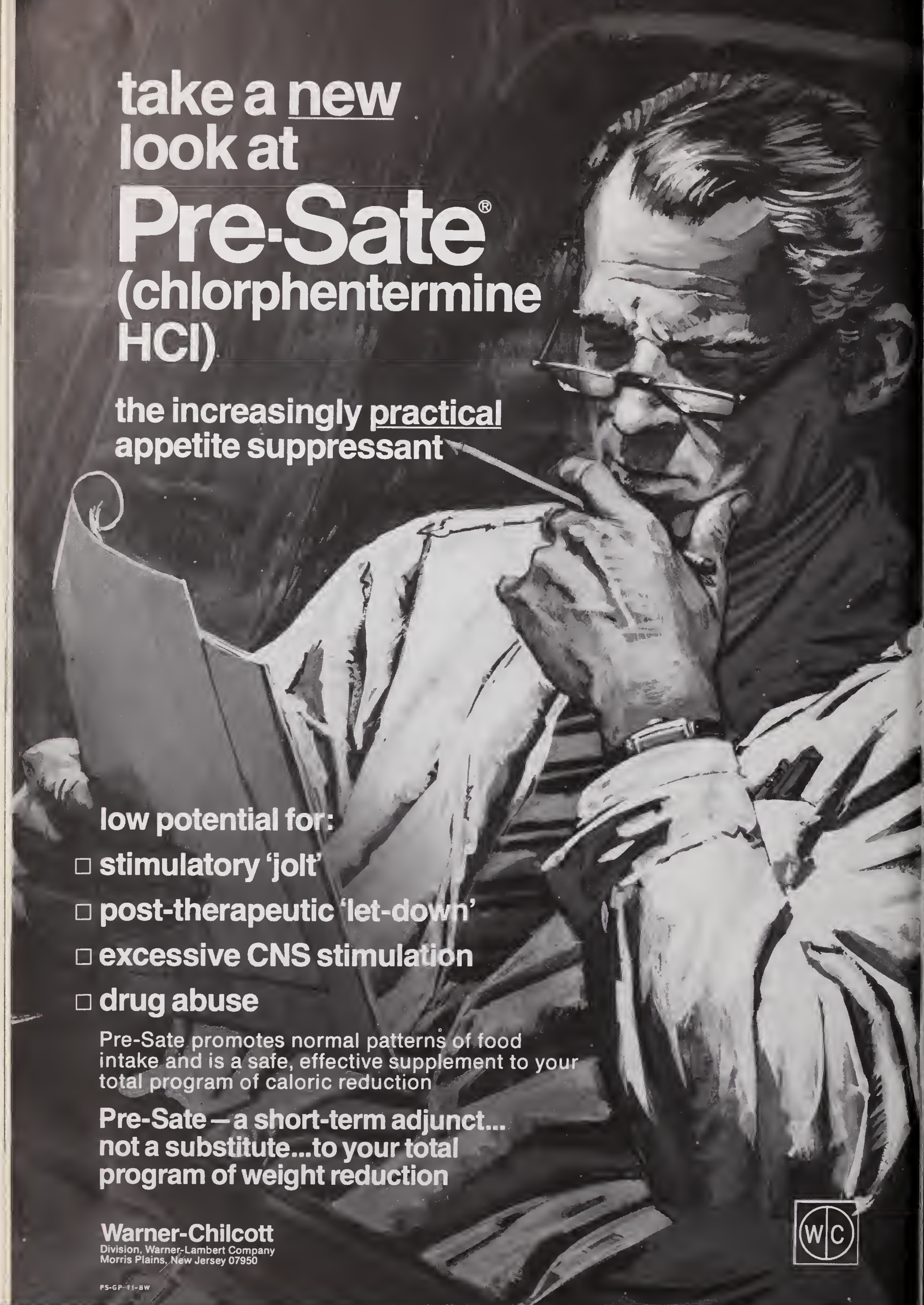

take a new look at

Pre-Sate®

(chlorphentermine HCl)

the increasingly practical appetite suppressant

low potential for:

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☐ post-therapeutic 'let-down'

☐ excessive CNS stimulation

☐ drug abuse

Pre-Sate promotes normal patterns of food intake and is a safe, effective supplement to your total program of caloric reduction

Pre-Sate—a short-term adjunct... not a substitute...to your total program of weight reduction

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PS-GP-11-BW

Pre-Sate® (chlorphentermine hydrochloride)

aution: Federal law prohibits dispensing without prescription.

ndications

re-Sate (chlorphentermine hydrochloride) is indicated in ogenous obesity, as a short term (i.e. several weeks) adjunct a regimen of weight reduction based upon caloric restriction.

ontraindications

laucoma, hyperthyroidism, pheochromocytoma, hypersentivity to sympathomimetic amines, and agitated states. Preate (chlorphentermine hydrochloride) is also contraindicated patients with a history of drug abuse or symptomatic cardioscular disease of the following types: advanced arterioclerosis, severe coronary artery disease, moderate to severe ypertension, or cardiac conduction abnormalities with danger f arrhythmias. The drug is also contraindicated during or ithin 14 days following administration of monamine oxidase hibitors, since hypertensive crises may result.

arnings

hen weight loss is unsatisfactory the recommended dosage hould not be increased in an attempt to obtain increased anoxigenic effect, discontinue the drug. Tolerance to the anorectic ffect may develop. Drowsiness or stimulation may occur and ay impair ability to engage in potentially hazardous activities uch as operating machinery, driving a motor vehicle, or perrming tasks requiring precision work or critical judgment. herefore, such patients should be cautioned accordingly. aution must be exercised if Pre-Sate (chlorphentermine hydrohloride) is used concomitantly with other central nervous ystem stimulants. There have been reports of pulmonary hypernsion in patients who received related drugs.

rug Dependence Drugs of this type have a potential for abuse. atients have been known to increase the intake of drugs of is type to many times the dosages recommended. In longrm controlled studies with the high dosages of Pre-Sate, brupt cessation did not result in symptoms of withdrawal.

sage In Pregnancy The safety of Pre-Sate (chlorphentermine ydrochloride) in human pregnancy has not yet been clearly stablished. The use of anorectic agents by women who are or ho may become pregnant, and especially those in the first rimester of pregnancy, requires that the potential benefit be eighed against the possible hazard to mother and child. Use f the drug during lactation is not recommended. Mammalian eproductive and teratogenic studies with high multiples of the uman dose have been negative.

sage In Children Not recommended for use in children under 2 years of age.

recautions

n patients with diabetes mellitus there may be alteration of insulin requirements due to dietary restrictions and weight loss. Pre-Sate (chlorphentermine hydrochloride) should be used with caution when obesity complicates the management of patients with mild to moderate cardiovascular disease or diabetes mellitus, and only when dietary restriction alone has been unsuccessful in achieving desired weight reduction. In prescribing this drug for obese patients in whom it is undesirable to introduce CNS stimulation or pressor effect, the physician should be alert to the individual who may be overly sensitive to this drug. Psychologic disturbances have been reported in patients who concomitantly receive an anorectic agent and a restrictive dietary regimen.

Adverse Reactions

Central Nervous System: When CNS side effects occur, they are most often manifested as drowsiness or sedation or overstimulation and restlessness. Insomnia, dizziness, headache, euphoria, dysphoria, and tremor may also occur. Psychotic episodes, although rare, have been noted even at recommended doses. **Cardiovascular:** tachycardia, palpitation, elevation of blood pressure. **Gastrointestinal:** nausea and vomiting, diarrhea, unpleasant taste, constipation. **Endocrine:** changes in libido, impotence. **Autonomic:** dryness of mouth, sweating, mydriasis. **Allergic:** urticaria. **Genitourinary:** diuresis and, rarely, difficulty in initiating micturition. **Others:** Paresthesias, sural spasms.

Dosage and Administration

The recommended adult daily dose of Pre-Sate (chlorphentermine hydrochloride) is one tablet (equivalent to 65 mg chlorphentermine base) taken after the first meal of the day. Use in children under 12 not recommended.

Overdosage

Manifestations: Restlessness, confusion, assaultiveness, hallucinations, panic states, and hyperpyrexia may be manifestations of acute intoxication with anorectic agents. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension, or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Fatal poisoning usually terminates in convulsions and coma.

Management: Management of acute intoxication with sympathomimetic amines is largely symptomatic and supportive and often includes sedation with a barbiturate. If hypertension is marked, the use of a nitrate or rapidly acting alpha-receptor blocking agent should be considered. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendations in this regard.

How Supplied

Each Pre-Sate (chlorphentermine hydrochloride) tablet contains the equivalent of 65 mg chlorphentermine base; bottles of 100 and 1000 tablets.

Full information is available on request.

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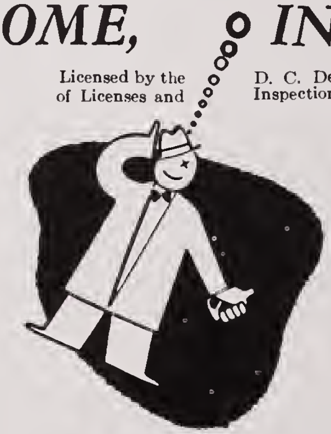

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brand of

pentazocine (as hydrochloride)

the long-range analgesic

a new outlook in chronic pain

of moderate to severe intensity

Contraindications: Talwin, brand of pentazocine (as hydrochloride), should not be administered to patients who are hypersensitive to it.

Warnings: *Head Injury and Increased Intracranial Pressure.* The respiratory depressant effects of Talwin and its potential for elevating cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions, or a preexisting increase in intracranial pressure. Furthermore, Talwin can produce effects which may obscure the clinical course of patients with head injuries. In such patients, Talwin must be used with extreme caution and only if its use is deemed essential.

Usage in Pregnancy. Safe use of Talwin during pregnancy (other than labor) has not been established. Animal reproduction studies have not demonstrated teratogenic or embryotoxic effects. However, Talwin should be administered to pregnant patients (other than labor) only when, in the judgment of the physician, the potential benefits outweigh the possible hazards. Patients receiving Talwin during labor have experienced no adverse effects other than those that occur with commonly used analgesics. Talwin should be used with caution in women delivering premature infants.

Drug Dependence. There have been instances of psychological and physical dependence on parenteral Talwin in patients with a history of drug abuse and, rarely, in patients without such a history. Abrupt discontinuance following the extended use of parenteral Talwin has resulted in withdrawal symptoms. There have been a few reports of dependence and of withdrawal symptoms with orally administered Talwin. Patients with a history of drug dependence should be under close supervision while receiving Talwin orally.

In prescribing Talwin for chronic use, the physician should take precautions to avoid increases in dose by the patient and to prevent the use of the drug in anticipation of pain rather than for the relief of pain.

Acute CNS Manifestations. Patients receiving therapeutic doses of Talwin have experienced, in rare instances, hallucinations (usually visual), disorientation, and confusion which have cleared spontaneously within a period of hours. The mechanism of this reaction is not known. Such patients should be very closely observed and vital signs checked. If the drug is reinstituted it should be done with caution since the acute CNS manifestations may recur.

Usage in Children. Because clinical experience in children under 12 years of age is limited, administration of Talwin in this age group is not recommended.

Ambulatory Patients. Since sedation, dizziness, and occasional euphoria have been noted, ambulatory patients should be warned not to operate machinery, drive cars, or unnecessarily expose themselves to hazards.

Precautions: *Certain Respiratory Conditions.* Although respiratory depression has rarely been reported after oral administration of Talwin, the drug should be administered with caution to patients with respiratory depression from any cause, severe bronchial asthma and other obstructive respiratory conditions, or cyanosis.

Impaired Renal or Hepatic Function. Decreased metabolism of the drug by the liver in extensive liver disease may predispose to accentuation of side effects. Although laboratory tests have not indicated that Talwin causes or increases renal or hepatic impairment, the drug should be administered with caution to patients with such impairment.

Myocardial Infarction. As with all drugs, Talwin should be used with caution in patients with myocardial infarction who have nausea or vomiting.

Biliary Surgery. Until further experience is gained with the effects of Talwin on the sphincter of Oddi, the drug should be used wi caution in patients about to undergo surgery of the biliary trac

Patients Receiving Narcotics. Talwin is a mild narcotic antagonis Some patients previously receiving narcotics have experienced mi withdrawal symptoms after receiving Talwin.

CNS Effect. Caution should be used when Talwin is administer to patients prone to seizures; seizures have occurred in a few su patients in association with the use of Talwin although no cause a effect relationship has been established.

Adverse Reactions: Reactions reported after oral administrati of Talwin include *gastrointestinal:* nausea, vomiting; infrequent constipation; and rarely abdominal distress, anorexia, diarrhe *CNS effects:* dizziness, lightheadedness, sedation, euphoria, hea ache; infrequently weakness, disturbed dreams, insomnia, synco visual blurring and focusing difficulty, hallucinations (see *Ac CNS Manifestations* under WARNINGS); and rarely tremor, ir tability, excitement, tinnitus. *Autonomic:* sweating; infrequent flushing; and rarely chills. *Allergic:* infrequently rash; and rare urticaria, edema of the face. *Cardiovascular:* infrequently decrea in blood pressure, tachycardia. *Other:* rarely respiratory depressio urinary retention.

Dosage and Administration: *Adults.* The usual initial adult dose 1 tablet (50 mg.) every three or four hours. This may be increas to 2 tablets (100 mg.) when needed. Total daily dosage should n exceed 600 mg.

When antiinflammatory or antipyretic effects are desired in ad tion to analgesia, aspirin can be administered concomitantly wi Talwin.

Children Under 12 Years of Age. Since clinical experience in ch dren under 12 years of age is limited, administration of Talwin this age group is not recommended.

Duration of Therapy. Patients with chronic pain who have receiv Talwin orally for prolonged periods have not experienced wit drawal symptoms even when administration was abruptly disco tinued (see WARNINGS). No tolerance to the analgesic effect h been observed. Laboratory tests of blood and urine and of liver a kidney function have revealed no significant abnormalities aft prolonged administration of Talwin.

Overdosage: *Manifestations.* Clinical experience with Talwin ove dosage has been insufficient to define the signs of this condition.

Treatment. Oxygen, intravenous fluids, vasopressors, and oth supportive measures should be employed as indicated. Assisted controlled ventilation should also be considered. Although nalo phine and levallorphan are *not* effective antidotes for respirato depression due to overdosage or unusual sensitivity to Talwin, pa enteral naloxone (Narcan®, available through Endo Laboratories) a specific and effective antagonist. If naloxone is not available, pa enteral administration of the analeptic, methylphenidate (Ritalin® may be of value if respiratory depression occurs.

Talwin is not subject to narcotic controls.

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Effect on mental acuity: Usually minimal on proper maintenance dosage. (See Warnings in summary of prescribing information.)

Safety: An excellent clinical record. In general use, the most common side effects reported have been drowsiness, ataxia, and confusion, particularly in the elderly and debilitated.

Concomitant use: Is used as adjunctive antianxiety therapy concomitantly with certain specific medications of other classes of drugs, such a cardiac glycosides, antihypertensive agents, diuretics, anticoagulants, anticholinergics and antacids. Although clinical studies have not estal lished a cause and effect relationship, physicians should be aware that variable effects on blood coagulation have been reported very rarely in patients receiving oral anticoagulants and chlordiazepoxide hydrochloride.

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(chlordiazepoxide HCl)

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up to 100 mg daily in severe anxiety

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Indicated when anxiety, tension and apprehension are significant components of the clinical profile.
Contraindications: Patients with known hypersensitivity to the drug.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported. Use of any drug in pregnancy, lactation, or in women of childbearing age requires that its potential benefits be weighed against its possible hazards.
Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.
Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported Also encountered are isolated instances c skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased anc decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltag fast activity) may appear during and afte treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.
Supplied: Librium capsules containing 5 mg, 10 mg or 25 mg chlordiazepoxide HCl. Libritabs® tablets containing 5 mg, 10 mg or 25 mg chlordiazepoxide.

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volume 41 / number 5 / may, 1972

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PRESIDENT'S MESSAGE

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Darrell C. Crain, M.D.

THE BENNETT AMENDMENT

I hope every member of our Society will read this page to the end. It is not an editorial nor the usual "President's Page." It is rather a statement of some facts which are not generally known, but with which every practicing physician must become familiar.

Senator Wallace F. Bennett (R-Utah) has introduced an amendment to H.R. 1 (the Social Security Act of 1971) which has become known as "The Bennett Amendment" and which deals with Professional Standards Review Organizations (PSRO's). This amendment, which has already passed the Senate Finance Committee and which Senator Bennett confidently predicts will become law profoundly affects - and to some degree alters - the practice of medicine as we know it today. Basically, the act calls for peer review in its broadest form - the actual continuous monitoring of every doctor's practice - quality of care provided, utilization of services, fees charged, etc. Similar "supervision" of hospital activities is to be performed.

To accomplish this, the Secretary of Health, Education and Welfare is instructed not later than January 1, 1973 to enter into an agreement with a "qualified organization whereby such an organization shall be designated as the Professional Standards Review Organization for that area." The "qualified organization" may be: a) a nonprofit professional association composed of licensed doctors of medicine or osteopathy, or b) such other public, nonprofit private or other agency or organization which the Secretary determines to be of professional competence.

To your officers the message is quite clear: we must move immediately to establish an organization that meets the qualifications specified. But like most problems today, the final solution is not simple. The organization must be open to all doctors of medicine and osteopathy in the area without regard to their membership in any other medical society or organization. This requirement appears to be best met through the establishment of a Medical Foundation. Fortunately, a committee under the chairmanship of Dr. James Boland has been studying this subject for more than a year. Recently, officers of your Society and chairmen of the Utilization and Peer Review and Fee Review Committees met with Dr. Boland to outline preliminary plans for such a Foundation. Discussions have been held with our legal counsel, and incorporation papers

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will shortly be signed. After that the membership will be circulated and each physician given an opportunity to join. Although the Act states that membership is to be on a voluntary basis, the following facts would seem to make membership mandatory: 1) The PSRO will not only review individual cases, it will actually monitor the quality of all physicians' practices, so all participating physicians will have a voice in setting standards. 2) All members will participate in the review process, as this must be on a rotating basis (for which a fee will be paid). 3) Insurance carriers will be obliged to deal with the PSRO on a contractual basis, thus giving the PSRO considerable influence in regard to their practices.

These facts are a brief summary of the most important features of the Bennett amendment. As soon as the basic organization is complete a printed summary will be prepared and mailed to the membership. The officers of the Medical Society of the District of Columbia intend to do all in their power to make sure physicians control their own destiny.

Darrell C. Crain

EdiTORiAls

SMOKING, YOU AND YOUR HOSPITAL

At any given time in any busy general hospital one could easily find a patient scheduled for a thoracotomy for bronchogenic carcinoma, another on the schedule for a laryngectomy, others visiting radiotherapy for treatment of advanced states of both of the above diseases and others on the medical wards for the treatment of the terminal stages of airway carcinoma. In addition, the intensive care unit will probably harbor at least one victim of respiratory failure due to bronchitis and/or emphysema.

The hospitals are fulfilling their missions by providing care for patients with these diseases. However, hospitals have health education as part of their role and in some instances locally that mission is not being fulfilled.

For a hospital to treat smoking associated diseases and yet permit the sale of cigarettes on its premises is hypocritical. For a medical staff to fail to take a position on this issue is for them to abdicate their role as doctors, in the true etymological sense of that word.

The following listing documents the status of the sale of cigarettes at local non-governmental hospitals based on a telephone survey to their administrative offices as of the middle of April.

CIGARETTES SOLD

- Columbia
- Children's
- George Washington
- Northern Virginia Doctors
- Sibley
- Suburban
- Washington Hospital Center

CIGARETTES NOT SOLD

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- Arlington
- Cafritz
- Casualty
- Doctors
- Fairfax
- Freedmen's
- Georgetown
- Hadley
- Holy Cross
- Jefferson
- National Orthopedic
- Prince Georges
- Providence
- Washington Sanitarium

Many of the hospitals where cigarettes are not sold have taken this action in the last year or two at the instigation of their medical staffs. As responsible, aware and involved physicians, each of us should work for the elimination of the sale of cigarettes in our own hospitals.

Robert Langevin, M.D.

MEDICAL SERVICE OF THE DISTRICT OF COLUMBIA

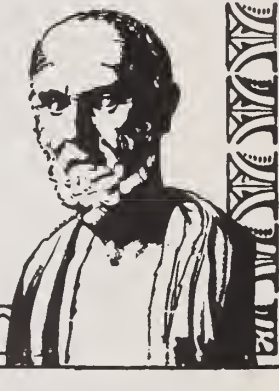
The growth of Medical Service of D.C. (Blue Shield) from a small "doctors plan" designed to provide covered services for low income patients to its present UCR plan for Federal employees has been nothing short of phenomenal. Recent events suggest that this growth may be accompanied by some disregard for the participating physician, without whom the plan could not exist.

The publication in the Washington Post of the amount of money paid to several physicians, naming these physicians, only serves to undermine public confidence in all physicians. It casts an unwarranted slur on the physicians involved before they are even accused of any wrongdoing, much less convicted. This type of guilt by inference and innuendo has no place in honest journalism and the Washington Post's actions are to be severely criticized.

Several months ago Mr. Victor Brian, President of Medical Service, was apparently quoted out of context in the Washington Post about physician overcharges. He subsequently apologized to the participating physicians for this inadvertent "leak." In the wake of this apology we read the recent "disclosure" in the Washington Post. Would not the interests of all concerned be better served if appropriate investigation preceded any statements to the press? It seems to us that the present tactics further weaken the confidence of the participating physicians in the management of Medical Service.

It is time for the participating physicians to begin asking some questions themselves. We have an opportunity to make ourselves heard at the annual meeting in June. Every participating physician should attend.

Jerome Canter, M.D.



"Healing is a matter of time, but sometimes also of opportunity."

Hippocrates, *Precepts*, Chapt. I

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Current Status of Exchange Transfusion in Fulminant Hepatitis

RAFAEL A. RIVERA, M.D.*

Under the best of modern-care facilities conventional treatment of acute hepatitis in coma carries with it a 90% mortality.[1] Intensive supportive care is mandatory, and corticosteroids are usually given in spite of little data to show their worth. The clinical course is usually catastrophic. It is characteristically attended or complicated by seizure disorders, cardiorespiratory aberrations, acid-base imbalance, coagulation deficiencies, gastrointestinal bleeding, and finally oliguria, ominously signalling a prompt terminus. Only an occasional patient will last longer than 2 weeks from the onset of coma.[2]

In 1966 two simultaneous reports[3,4] relating the benefits of exchange transfusion in fulminant hepatitis initiated renewed enthusiasm for a procedure that had been sporadically used in the past to treat acute hepatitis in coma.[5-7] During the suceeding 5 years 103 patients[8,9] so treated have been formally introduced into the literature with an encouraging 32.0% recovery rate. Other observers[10] maintain a skeptical view, and still others have suggested better survival without than with the use of exchange transfusion.[11] Increasing interest resulted in the establishment of the Fulminant Hepatic Failure Surveillance Study group located in Boston under the direction of Doctor Charles Trey, author of a report of the first 12 cases of fulminant hepatitis treated by exchange transfusion reported in North American literature. Case histories are sent in from all over the United States, regardless of form of treatment, to be gathered and analyzed in this central point. The results of therapy in the first 284 cases culled by the FHFSS[1] are shown in table I.

As shown, the use of exchange transfusion with or without steroids doubled the survival obtained with conservative therapy with or without steroids. Studies are currently in progress blindly randomizing patients to conservative management versus therapy with exchange transfusion. With few exceptions most observers think that a controlled trial is required to establish the therapeutic value of this procedure, and more so when it has been suggested that this complication of hepatitis is probably on the increase.[9] The literature does not reflect the true incidence of fulminancy. An extensive 18-year literature survey[8] uncovered 263 patients, whereas the Fulminant Hepatic Failure Surveillance Study was able to gather 284 patients in less than 3 years. Physicians are reluctant to publish reports of 1 or 2 cases of fatal fulminant hepatitis for the sake of interest alone without relating any new contributions. The vast majority of cases pursue a montonously similar, disastrous, short-lived course that ends fatally and fails to be recognized. The following observations combine my experience with 12 such patients treated with exchange transfusion.

What are the indications for exchange transfusion in fulminant hepatitis? No one single factor or combination of factors determines the need for an exchange transfusion once the patient reaches coma. No improvement after 24 hours of intensive conventional management to include the use of corticosteroids is a suitable waiting period prior to initiation of the procedure. In essence, this means that once the patient is in coma,

TABLE I: Results of Therapy in 284 Patients with Fulminant Hepatitis

Therapy	Per Cent Survival
Conservative treatment	7.1
Conservative treatment plus steroids	11.0
Exchange transfusion	23.8
Exchange transfusion plus steroids	25.7
Other procedures*	8.3

* Hemodialysis, heterologous liver perfusion, cross circulation.

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preparations are started, blood requested, cannulas inserted, and consultations obtained, all of which usually take a day to complete. Clearly the patient should have reached full unconsciousness prior to considering the procedure, since lighter stages of encephalopathy are usually attended by seizure disorder, agitation, uncontrollable movements and thrashing about, all of which preclude the satisfactory conduct of an exchange transfusion.

Even though it has been tried, there is no enthusiasm for exchange transfusion in the decompensated cirrhotic or other forms of end-stage, chronic liver disease with coma.[12] In these cases coma is usually acutely reverted, only to be followed by relapses and no lasting influence on the clinical course.

How is an exchange transfusion done? The simplest indirect way is to alternatively infuse and withdraw blood through a large-bore cannula inserted into the inferior vena cava, allowing a few minutes for equilibration between influx and efflux. Blood may also be administered into a large vein, e.g., the inferior cava, and withdrawn sequentially or simultaneously from a free-flowing artery such as the radial into a sterile collection bag. A direct exchange means that donors are brought to the bedside and connected to the patient, usually with the help of a pump to assist undirectional flow from the donor into the recipient patient. In the plasmaphoresis (plasma exchange) method, blood withdrawn from the patient is centrifuged at the bedside, red blood cells are separated from plasma, reconstituted with fresh frozen plasma, and returned to the patient. Commercially available blood-cell separators do all this—withdrawal, centrifugation, and re-infusion of the patient's own formed elements, automatically and at a faster predeterminable rate.

How much blood to infuse or withdraw at a given time is arbitrary. Whole units or 500 cc aliquots seems to be a reasonable and convenient figure. The total blood volume needed to replace can also vary. It has been calculated that a 10-unit whole-blood exchange replaces 80% of the patient's initial blood volume.[13]

An uncomplicated 10-unit exchange is usually completed in 3 to 5 hours. The plasma-exchange method may take longer, depending on the speed of the bedside centrifuge being used and its braking ability. All effluent blood should be placed in large plastic bags and incinerated. Involved personnel should wear gloves and use exceptional care to prevent transmission of hepatitis virus.

Once the exchange transfusion is in progress, how and when does improvement come about? Most survivors awaken—if they are going to do so—with 1 or 2 exchange transfusions. Only 3 patients have recovered after 3 exchanges, none after 4, 5 or 6 and only 2 after 7 exchanges (1 of which was cross circulated to a baboon). Improvement comes about slowly; varying neurologic signs are observed, ranging from lightening in the state of consciousness, swallowing, purposeless body and eye movements, all the way to full alertness. Oscillations in the level of consciousness are common.

In the absence of neurologic recovery are there any solid indicators of improvements? Unfortunately not. A 10-unit exchange replacing approximately 80% of the patient's initial blood volume artifically lowers and raises serum chemistries. A continuing rise in bilirubin with a concomitant continued fall in transaminase indicates further reduction in functioning liver-cell mass. The serum ammonia level, so often referred to, bears no relation to the level of consciousness. An equally poor correlation with the state of consciousness has been noted in serially obtained and graded electroencephalograms in patients treated via exchange transfusion.[12] Furthermore, adequate EEG tracings are difficult to obtain because of electrical interference from mechanical respirators so often required to assist ventilation and muscle artifacts from decerebrate postures, blinking of eyes, and involuntary motions of the head and body.

A note of caution is in order on the use of the electroencephalograms as an indicator of "cerebral death". The absence of electrical activity in the brain cannot be interpreted as an unequivocal sign of death unless it persists for over 24 hours;[14] recovery after a "flat EEG" has been reported.[15]

Is there any relation between the etiology of the underlying hepatitis and eventual prognosis? Most cases of fulminant hepatic necrosis are due to viral hepatitis followed by toxic or drug-related acute liver injury. The survival of the latter group is lower than that of patients with viral hepatitis, the largest toll in this group being taken by Halothane-associated cases. Of these, only 1 in 13 patients has been officially described in the literature as a recovery through exchange transfusions. Data from the Fulminant Hepatic Failure Surveillance Study has repeatedly indicated that Halothane was the second most common associated cause of massive liver-cell necrosis.[1,16] One patient with hepatic failure in the third trimester of pregnancy recovered after exchange transfusion.[13]

If the patient awakens from coma, what does this mean in relation to long-term survival? The literature does not permit clear assessment of how often patients fully regain consciousness while being treated conventionally, regardless of eventual outcome. Presumably this happens only occasionally. On the other hand, several patients have been reported who recovered from coma after exchange transfusion but subsequently succumbed to complications such as convulsions, severe bronchospasm, coagulation abnormalitities, and severe bleeding as long as 5 weeks after return of consciousness. In dealing with the scanty data available it seems that awakening from coma can be expected oftener in association with than without an exchange transfusion. Even though neurologic recovery does not necessarily guarantee eventual survival, it may increase its likelihood.

What are the indications to cease exchange transfusion? There are no clinical observations, biochemical determinations, or electroencephalographic interpretations that can be used prognostically. Perhaps histologic confirmation of liver-cell regeneration might justify continued therapy when clinical signs of recovery are not forthcoming. Unfortunately, coagulation abnormalities in these patients prohibit liver biopsy. Some observers have indicated their willingness to pursue further exchanges as long as blood was available and it was otherwise feasible.[17] Sherlock[18], however, thinks that the procedure should be abandoned if no improvement ensues after 2 exchanges spaced 12 hours apart followed by 2 additional exchanges at 24-hour intervals. Five of ence with the procedure will certainly first or second exchange transfusion, and this has been the experience of most observers. Existing facilities, availability of blood, and prior experience with the procedure will certainly enter into consideration. In the absence of factual data, any decisions on the duration of therapy in the absence of neurologic improvement remain arbitrary.

What is the rationale for exchange transfusion? There is no answer now. Two general speculations are proposed: one is the removal of some unknown toxic substance (s), and the other is the removal of some unknown material (s) that promotes recovery. To be sure, ammonia and bilirubin can be rapidly removed in great amounts with only variable and inconsistent effects on the patient's status. If exchange transfusion proves to be more helpful than hemodialysis through the removal of a toxic substance only 1 conclusion can be reached—whatever the substance is, it must be protein-bound. Af first glance it appears virtually impossible to successfully search and identify a protein-bound substance in itself capable of producing irreversible coma in experimentally-induced animal hepatitis.

What initially appeared to be the weaker of the 2 speculations—the possible introduction of a healing factor—has gained stronger support. In experimentally-induced canine massive liver-cell necrosis, exchange transfusion with blood high in canine hepatitis virus-antibody resulted in recovery of most dogs exchanged with non-immune blood.[19] Further repetition with susceptible animals who received 5 cc of hyperimmune serum and no exchange transfusion resulted in recovery of all dogs, but not others thus demonstrating antibody-dependent recovery in the experimental animal. On the other hand, beneficial effects of exchange transfusion have been noted in carbon tetrachloride-induced hepatic necrosis in monkeys, a situation wherein the role of antibody does not appear to be a factor.[20]

The most impressive observation in humans has been the recovery of 5 out of 8 patients with fulminant hepatitis who were given 1 to 2 units of plasma from Australia antigen-positive hemophiliacs hyperimmunized by repeated transfusion.[21] This donor plasma had a titer of 1:4 to 1:8 as measured by gel immunodiffusion. Unfortunately, some patients received steroids and others exchange transfusion as well. To resolve the dilemma, a randomized double-blind study is being conducted in which some patients are being given Australia antigen-antibody in the form of hyperimmune gamma globulin and others ordinary gamma globulin.

What is a successful exchange transfusion? The answer to this question is not obvious. The general purpose of an exchange transfusion is to sustain life long enough for liver-cell regeneration to occur. Hopefully, the patient will recover from coma, continue to improve, leave the hospital, and eventually return to his former occupation. However, as I have pointed out, awakening from coma does not necessarily insure long-term survival; relapses into coma or other complications leading to demise occur all too often.

Let us consider for a moment the patient who recovers after an exchange transfusion and continues to improve but convulses and dies a month later. Was that a successful exchange transfusion or not? An 18-year-old girl awakened after a second exchange and convalesced through full ambulation to tolerate a regular diet, and then bled torrentially to death from a severe hemorrhagic gastritis with underlying familial vascular telengiectases. Was that a successful exchange transfusion? Too many patients recover from coma and eventually die from seemingly far-removed complications at a time when liver-cell regeneration, as determined at autopsy, and liver function would have been sufficient to easily sustain life.

To properly approach this question of transient neurologic reversal and its relation to long-term recovery as a measure of therapeutic success, one falls back once more on the question dealt with before: how many patients treated conventionally regain consciousness spontaneously, and of these how many die of unrelated causes? Unfortunately, there is not enough information available in the literature to obtain a satisfactory answer. It is reasonable to assume that spontaneous recovery from coma occurs only in a minority of patients. The clinical course is so fulminant that death usually occurs in a few days, some patients lasting a week, occasionally 2, but will hardly ever spontaneously regain full consciousness. For this reason I personally feel that full neurologic recovery is a rare event, much to be hoped for and rarely forthcoming, which should be considered the initial aim of therapy. Whether this in turn will be followed by an uncomplicated course thereafter remains to be seen. Certainly hemodialysis is not considered a failure when azotemia is reverted but the patient proceeds to die from renal failure.

Summary

Exchange transfusion in the treatment of acute hepatitis in coma offers some promise in an otherwise usually hopeless situation. Currently available information indicates a doubling of survival figures with the use of exchange transfusion as opposed to conventional therapy with corticosteroids. If facilities are available, exchange transfusion should be considered if improvement is not forthcoming while under usual intensive supportive care.

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Calcium Transport by Isolated Cardiac Sarcolemma*

CONSTANTINOS J. LIMAS, M.D.†

A substantial body of evidence accumulated during the past few years ascribes to the calcium ion the unique role of the immediate stimulus to contraction in both skeletal muscle and heart.[1-4] It is now accepted that the role of calcium in this respect is indirect in that it initiates contraction by reversing the inhibition exerted by the troponin-tropomyosin system on the interaction of actin and myosin.[5] Relaxation of the myofibrils, according to this schema, requires the removal of Ca^{++} from the contractile apparatus.

Since rapid, reversible changes in the concentration of calcium ions in the myoplasm are critical to the maintenance of normal contraction-relaxation cycle, regulatory mechanisms ensuring intracellular Ca^{+} levels appropriate for each phase of the contraction sequence are required. Experimental evidence has shown that the endoplasmic reticulum and, to a lesser degree, mitochondria fulfill this regulatory role by virtue of their capacity to sequester calcium against large concentration gradients. Because it occurs against a concentration gradient. Ca^{++} uptake by these organelles requires the expenditure of energy supplied by ATP. Both Ca^{++}-activated and Na+K-activated ATPases have been identified in sarcoplasmic reticulum and mitochondria from skeletal muscle and heart.[6,7]

Calcium seems to be released from the sarcoplasmic reticulum not only following depolarization but also in response to pharmacological influences (ouabain,[8,9] caffeine,[10] epinephrine[11]). On the other hand, the depressed contractility of heart failure, whether spontaneous or experimentally induced, is associated with a decreased calcium uptake by cardiac endoplasmic reticulum and mitochondria.[12-14]

Although the role of sarcoplasmic reticulum and mitochondria in intracellular and transmembrane Ca^{++} fluxes has been intensively studied, the contribution of the sarcolemma has not been clarified, mainly because of the difficulty in obtaining satisfactory preparations. Recently, reliable and reproducible methods for isolating sarcolemma were described.[15-17] The present study was, therefore, undertaken to define the role of sarcolemma in calcium transport.

Material and Methods:

Adult mongrel dogs were sacrificed and their hearts rapidly excised and placed in crushed ice for 30 min. The left ventricle was dissected free of epicardial fat and vessels and a 5-gm specimen obtained from the free ventricular wall. The specimen was homogenized for 45 sec, in a Waring blender. The homogenizing solution contained 0.3 M sucrose, 20 mM tris-HCl (pH 7.4) and 1 mM ethyleneglycoltetraacetate (EGTA); 5 mM sodium azide were added to the medium when isolation of the endoplasmic reticulum was desired. Further fractionation was obtained as follows:

Preparation of Sarcolemma.[16] The tissue homogenate was centrifuged at 2.500 X g for 20 min. The resultant sediment was suspended in the homogenizing solution and washed and spun at 4000 X g for 10 min. The procedure was repeated 6 times. The final sediment was obtained by centrifugation at low speed (450 X g) for 6 min. The residue obtained was extracted with 2 M NaI for 16 hrs in the cold, centrifuged at 5.000X g for 10 min., and washed twice with sucrose. It was finally suspended in the homogenizing medium and its protein content measured by the method of Lowry et al.[18]

Preparation of mitochondria and sarcoplasmic reticulum. The tissue homogenate was centrifuged at 1.000 X g for 20 min. to separate the cellular debris. The supernatant was subsequently centrifuged at 8.000 X g for 30 min. to obtain the mitochondrial pellet. The "pest-mitochondrial" supernatant was then spun at 80.000 X g for 1 hr to isolate the sarcoplasmic reticulum pellet. The composition of the preparations was confirmed in each instance by electron microscopy. The final pellets were suspended in the homogenizing medium to a protein concentration of 1 mg/ml.

Na+K-activited ATPase activity. This was determined by continuously monitoring the oxidation of NADH in a Beckman DU-2 recording spectrophotometer at a wavelength of 340 mu by means of a linked enzyme system.[19] Each cuvette contained 5 mM $MgCl_2$, 100 mM NaCl, 10 mM KCl, 25 mM tris-HCl (pH 7.4), 2.5 mM tris-ATP, 0.5 mM NADH, 2.5 mM phosphoenolpyruvate (PEP), and 0.02 ml of pyruvate kinase/lactate dehydrogenase suspension in a final volume of 1.9 ml. The reaction was started after temperature equilibration by the addition of 0.1 ml of the membrane suspension containing Na+K-ATPase. The ADP formed from ATP in the assay medium by the action of (Na+K)-ATPase forms, in the presence of PEP and pyruvate kinase, pyruvate and ATP. Pyruvate in the presence of NADH and lactate dehydrogenase then forms lactate and NAD. Since all additions in the assay medium, except for the ATPase, were in excess, the hydrolysis of ATP is the rate-limiting step in the reaction sequence, and the amount of NAD formed is directly proportional to the amount of ATP hydrolysed by the Na+K-activated ATPase. The ATPase activity in the absence of KCl and NaCl (*Mg^{++}-ATPase*) was also determined and substracted from the total (Na-K-Mg)-ATPase activity.

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Ca^{++}-activated ATPase activity. This was determined by incubating the tissue preparation in a medium containing 50 mM tris-HCl (pH 7.4), 1 mM EGTA, 4 mM ATP with or without 2 mM calcium. After 10 min. incubation the reaction was stopped with 10% trichloroacetic acid, and inorganic phosphorus was determined by the Fiske-Subbarow method.[20]

Rate of calcium uptake by the subcellular fragments. This was determined[21] in incubation mixtures of 10 ml containing 5 mM sodium oxalate, 5 mM $MgCl_2$, 5 mM ATP, 0.11 M NaCl, 0.01 M KCl, 0.01 M tris-HCl (pH 7.4), 1.5×10^{-3} M $CaCl_2$ and 1.2 mg vesicular or mitochondrial or sarcolemmal protein per ml. The incubation mixtures were drawn immediately into plastic syringes and, at appropriate time intervals, 2-ml aliquots were filtered through millipore filters (0.45 μ).

Calcium determinations were performed with a Perkin-Elmer model 290 atomic absorption spectrophotometer. All samples were assayed in aqueous solutions containing 0.4 M perchloric acid and 0.2 g/100 ml $LaCl_3$. Standard solutions of calcium carbonate were prepared, and these were linear and reproducible between 0.01 and 0.1×10^{-3} M. Standards with the tissue specimens were prepared after extraction of the subcellular fractions with 0.4 M $KClO_4$ containing 0.2 g/100 ml $LaCl_3$ for 30 min. and centrifugation to obtain the protein-free supernatant solution.

The effect of ouabain on calcium uptake by the sarcolemmal preparation was studied by adding 0.02 ml of the ouabain solution (in 3 different concentrations (5×10^{-7} M, 5×10^{-5} M, and 5×10^{-4} M) to the assay medium. The ionic composition of the incubation mixture was also varied by altering the ratio of NaCl to KCl. The following combination of concentrations was used: 10, 20, 40, 80mM NaCl, each in combination with 10, 20, 40 and 80 mM KCl. Calcium uptake was determined after 10 min. of incubation.

Calcium release by sarcolemma. This was studied after incubation of the preparation with graded concentrations (5×10^{-7} M, 5×10^{-5} M, 5×10^{-4} M) of ouabain and determination of Ca^{++} concentration in 2-ml aliquots filtered through 0.45 μ millipore filters. The incubation medium without added ouabain served as control.

Calcium transport by sarcolemma was also studied in "aged" preparations obtained by storage of the sacolemmal pellets in 20 ml of sucrose solution (0.25 M, pH 7.4) at –4° C. Calcium uptake and release studies were made after 1-4 days of storage..

Results

In the presence of ATP and oxalate, sarcolemmal preparations sequestered calcium ions to a lesser extent than mitochondria or sarcoplasmic reticulum. At a calcium concentration of 1.5×10^{-3} M, sarcolemma accumulated 0.32 μmole Ca^{++}/mg protein after 10 min. of incubation compared to 0.52 μmole Ca^{++}/mg protein for sarcoplasmic reticulum and 0.42 μmole Ca^{++}/mg protein for mitochondria (Fig. 1). Moreover, the rate of initial Ca^{+} uptake by sarcolemma was somewhat delayed in comparison to the other organelles.

Calcium uptake by cardiac sarcolemma fell to very low levels when the preparation was incubated in the abscence of ATP; removal of oxalate or incubation at 4° C also resulted in decreased uptake (Fig. 2). "Aging" of the sarcolemmal membranes was followed by a rapid and progressive loss of Ca^{++} uptake capacity. This was paralleled by a loss of Ca^{+}-ATPase activity, while (Na+K)-ATPase was little affected or actually increased somewhat (table 1).

The effect of varying the relationship of NaCl to KCl in the incubation medium on the uptake of calcium by sarcolemma was not statistically significant. Similarly, ouabain did not affect Ca^{++} uptake; calcium release by sarcolemma, however, was increased in the presence of ouabain. The ouabain-induced Ca^{++} release was proportional to the concentration of the drug in the assay medium. When "aged" preparations of sarcolemma were used the amount of Ca^{++} released was significantly less and there was no further increase with higher doses of ouabain (table 2).

(Na+K)-ATPase activity of sarcolemmal preparations was significantly higher than that of sarcoplasmic reticulum or mitochondria. It was inhibited by ouabain and depended on the presence of ATP in the assay medium. Ca^{++}-activated ATPase, on the other hand, was less active in sarcolemma than in sarcoplasmic reticulum or mitochondria (table 3).

Discussion

The results of the present study indicate that sarcolemma plays an important role in the regulation of intramyocardial Ca^{++} concentrations, thereby participating in the excitation-contraction coupling mechanisms. In a way analogous to other organelles studied before (sarcotubular vesicles and mitochondria) sarcolemma from the dog heart can sequester calcium and can be induced to release this ion in response to low doses of ouabain. This Ca^{++} uptake is an energy-linked process, since omission of ATP from the incubation medium leads to a dramatic fall in the amount of Ca^{++} accumulated by sarcolemma. The presence of both Na+K-dependent and Ca^{++}-activated ATPases in our sarcolemmal preparations confirm recent observations by other investigators[16,17] and may provide the enzymatic basis for calcium transport by sarcolemma.

The exact mechanisms by which membranes control the intracellular Ca^{++} concentrations at levels appropriate for the instantaneous phase of the contraction-relaxation cycle are not clearly understood. It seems, however, that these mechanisms are closely related to transmembrane fluxes of sodium and perhaps of other ions as well. A well-studied example is the activity of membrane Na+K-ATPase which is concerned with Na-K exchange.[21] This ubiquitous enzyme is, classically inhibited by ouabain at low concentrations (10^{-7} M), and is activated by external K$^+$ and inhibited by external Na$^+$. The Na-K exchange is not affected or is slightly increased by Ca^{++}-free media and increases in linear relation to intracellular Na concentration $[Na]_i$.

A different mechanism has been described in giant squid axons[21] and more recently in the heart via a Na-Ca

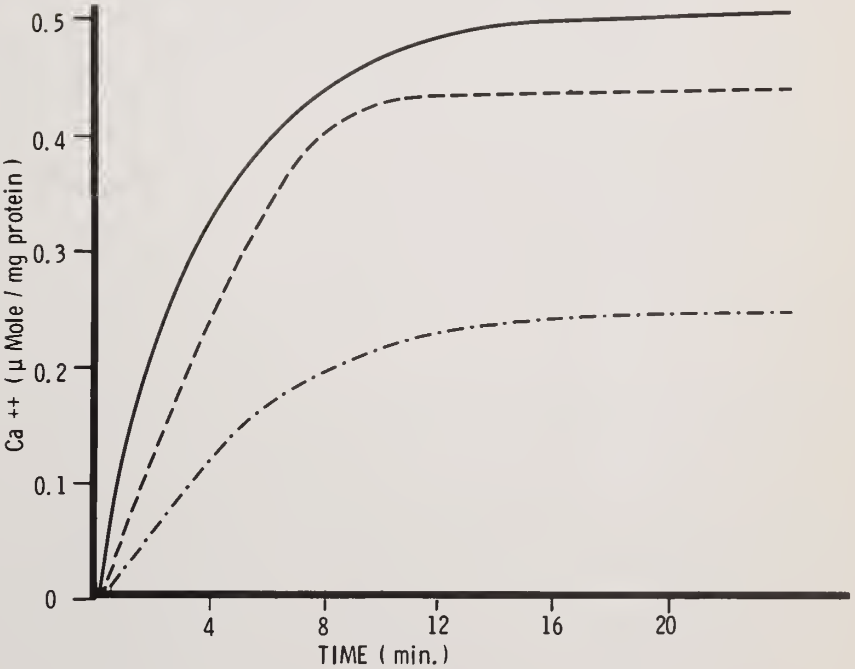

FIG. 1. Calcium uptake by sarcoplasmic reticulum (——), mitochondria (----) and sarcolemma (-·-·). Incubation mixture contains 5mM sodium oxalate, 5mM MgCl$_2$, 5mM ATP, 0.11 M NaCl, 0.01 M KCl, 0.01 M tris-HCl (pH 7.4), 1.5 × 10^{-3} M CaCl$_2$ and 1.2 mg protein/ml.

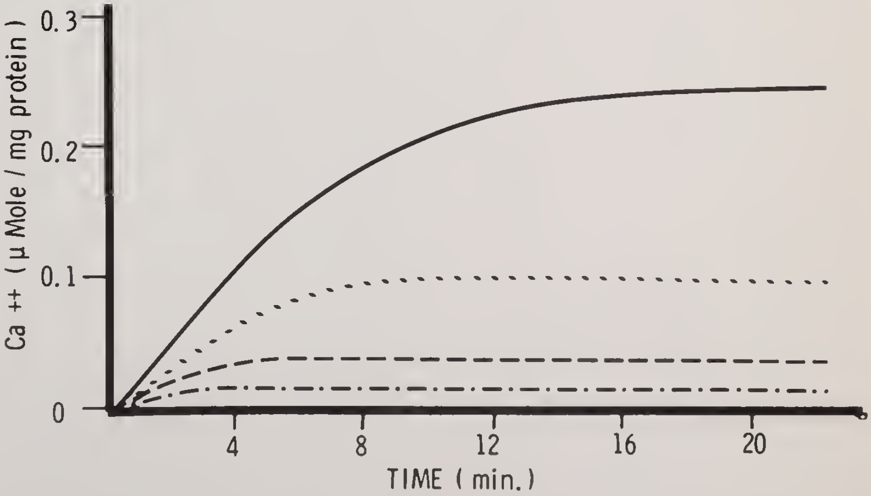

FIG. 2. Calcium uptake by dog heart sarcolemma. Control: (——) (incubation mixture as in FIG. 1. Uptake in the absence of oxalate (·····), at 4° C (----) and in the absence of ATP (-·-·).

TABLE 1: The Effect of "Aging" on Ca^{++} Uptake, (Na + K)-ATPase and Ca^{++}-ATPase of Dog Heart Sarcolemma

Duration of incubation (days)	Ca^{++} uptake (μmol/mg prot) 10 min. incubation	(Na + K)-ATPase (μmol P$_i$/mg prot) 1 hr incubation	Ca^{++}-ATPase (μmol P$_i$/mg prot) 1 hr incubation
Control	0.3 ± 0.02	16 ± 1.2	5 ± 1.0
1	0.07 ± 0.01	17 ± 0.9	4.8 ± 1.2
2	0.04 ± 0.01	15 ± 3.1	4.2 ± 0.8
3	0.03 ± 0.02	16.4 ± 2.5	3.7 ± 1.3
4	0.02 ± 0.01	18 ± 1.7	3.2 ± 0.7

TABLE 2: Effect of Ouabain on Ca^{++} Release by Fresh and "Aged" Sarcolemma

	Ca^{++} (μmol/mg prot/hr)	
Assay Medium	Fresh sarcolemma	Aged sarcolemma (5 days)
Control	3.2 ± 1.9	2.0 ± 0.8
5×10^{-7} M ouabain	3.9 ± 1	2.0 ± 1.2
5×10^{-5} M ouabain	4.2 ± 1.1	1.9 ± 1.1
5×10^{-4} M ouabain	4.2 ± 1.2	2.3 ± 0.7

exchange.[22] According to this mechanism, influx of Ca^{++} is linked to a high intracellular Na concentration. Indeed, Glitsch, Reuter and Scholz[22,23] observed an increase in Ca^{++} uptake by guinea-pig atria when the intracellular Na content was raised by various procedures. In view of the known extracellular Na-Ca antagonism for membrane-carrier systems, it would be tempting to speculate that a similar competition between Na and Ca also exists inside the membrane and that a rise in internal Na concentration would not only lead to an increase in calcium influx but also to a reduction of its efflux.

The Na-Ca exchange, in contrast to the Na-K exchange, is insensitive to even high doses (10^{-3} M) of ouabain. It is activated with low affinity by external K$^+$ (up to 100 mM) and Na$^+$, is roughly proportional to $[\mathrm{Na}]_i^2$, and is markedly reduced in Ca^{++}-free media and in the presence of EDTA or lanthanum.[22] Despite differences, the activities of these 2 mechanisms (Na-K and Na-Ca exchange) are closely interrelated. Inhibition of the Na,K-pump (e.g., by cardiotonic glycosides) would increase the intracellular Na concentration which, by activating the Na-Ca exchange, would lead to increased influx of Ca^{++}. Similarly, Davis and Vincenzi[24] have recently found a proportionately greater inhibition of the (Na+K)-ATPase (compared to the activation of Ca^{++}-ATPase) at Ca^{++} concentrations [10^{-7}] comparable to those found in the myoplasm during relaxation. This inhibition would augment or trigger the calcium influx for the next contraction-relaxation cycle. A third mechanism, in which internal Na is exchanged for external Na [the Na-Na pump], has been invoked in calcium transport by the squid axons,[25] but its role in muscle has not been defined.

Sarcolemma quantitatively is less active in a Ca^{++} uptake than sarcoplasmic reticulum or mitochondria in the dog heart. The net contribution of the organelles to Ca^{++} transport, however, depends on their quantitative relationship in the whole cell. It seems, from our data, that the role of sarcolemma in excitation-contraction coupling is supportive for the sarcotubular system which contains the bulk of the calcium storage sites.

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Screening the Negro Population for Inborn Errors of Metabolism

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In 1966, responding to the increased emphasis in screening newborns for those metabolic diseases that cause preventable mental retardation, a metabolic screening program was initiated at Howard Medical School in Freedmen's Hospital.

Initially, a pilot program was undertaken with personnel and equipment from the Medical Genetics Unit of the Department of Pediatrics. When equipment and techinical assistance were available, the program was expanded to include all infants born in Freedmen's Hospital. The results of this program from September 1965 to September 1971 are presented in this report.

Reasons for Screening

The majority of our States either now have laws or at least programs requiring screening for phenylketonuria (PKU), a disease causing preventable mental retardation. In addition to compliance with the law and for purely humanitarian reasons for screening newborns for these diseases, it costs the State, and ultimately the taxpayer, upwards of $100,000 for the lifetime care of an institutionalized mentally retarded individual.

Some controversy exists as to the scope and depth of a screening program. We believe that a program is justfied when the frequency of a disease in the population chosen is sufficiently high so that an adequate number of individuals benefit from the results or when the results would reveal a condition that, if untreated, would have dire consequences. Other general principles that apply to a consideration of any program have been outlined recently by Charles A. Janeway.[1]

1. The screening test must be simple, cheap, and easy to perform on small, readily collected stable samples.
2. The screeening must be oversensitive, so that all cases will be identified. There can be some false positives but no false negative tests.
3. There should be a satisfactory backup to the screening program so that positive tests can be corroborated by the use of more sensitive methods.
4. There should be an effective method of treatment for these disorders that will benefit the patient.

The latter principle is not always possible to adhere to because there are not effective methods of treatment for many of these disorders. However, it is important to identify these individuals and attempt treatment with classical methods of diet for the aminoacidopathies and galactosemia, avoidance of certain drugs with glucose-6-phosphate dehydrogenase (G6PD) deficiency or genetic counseling for the hemoglobinopathies.

In the Negro population, inherited disorders with a relative high frequency should be screened; these include the hemoglobinopathies and G6PD deficiency.[2] Others can be added when a biochemical procedure that follows the outlined general principles is correlated with a particular disease. Those inherited disorders with relatively low frequency in the Negro include cystic fibrosis, hemophilia, phenylketonuria (PKU), and Wilson's disease.

Material and Methods

Blood amino acids and enzymes are screened from blood obtained by a heelprick in the nursery and deposited on S&S 903 filter paper where it is dried. It is stable in this from and can be mailed to the laboratory as a followup specimen. A blood spot from each infant is screeened for G6PD deficiency by a method similar to Beutler's fluorescent procedure.[3] Any low values of enzyme activity are corroborated by the standard assay method.[4]

Blood spots for the G6PD assay can be stored dry over a desiccant at 4°C for several months with little or no loss of enzyme activity. We have found that specimens sent through the mail even during the summer months have arrived at the laboratory with a normal level of G6PD enzyme activity. Amino acids are evaluated by the Efron Technique which is a one-dimensional paper chromatographic multi-dip procedure.[5] This simple technique loses some sensitivity as compared with the

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Guthrie inhibition assay.[6] but in 1 operation we can see imbalances in most of the amino acids which are present in concentrations of above 4-6 mg/100 ml blood depending on the amino acid.[7] The somewhat cluttered area of the chromatogram around glycine-serine can be confusing occasionally, but the blood spots from these specimens are extracted with 65% ethanol and chromatographed in thin-layer (TLC) with the appropriate solvent system.[8] This screening procedure for amino acids is backed up by a Technicon TSM, automatic amino-acid analyzer with a computer attachment.

Both blood and urine specimens on filter paper are requested from the infant 1 month after the initial discharge from the nursery. Urine specimens are screened for amino acids by the chromatographic procedure. Samples that smear due to an occasional high salt content of the urine are extracted with 65% ethanol and checked by TLC or high voltage electrophoresis.[8] The urine spot on filter paper can be screened for cystine and homocystine by the sodium nitroprusside reaction.[9,10] This test is carried out on a white porcelain spot plate. Samples that are positive are re-assayed by the silver nitroprusside method that differentiates between cystine and homocystine.[11] The urine spots can be evaluated for mucopolysaccharides by the toluidine blue method.[9,10] Tests such as color, odor, keto acids, and reducing substances require fresh urine because this information cannot be obtained from followup specimens mailed into the laboratory. When fresh urine is available from the newborns or from high-risk patients, the sample can be subjected to a complete battery of tests including quantitative sugars, organic acids, imidazoles, phenolic acids, specific gravity, protein, and drug metabolites.[8-10] Generally we do not use the ferric chloride test for detection of keto acids because it is only a very preliminary test. Instead, we use the 2 , 4 dinitrophenylhydrazone procedure with subsequent TLC of the keto-acid complex.

Results

Table 1 shows the results from 11,919 Negro infants screened during the period September, 1965 to September, 1971. The numbers for amino acid do not indicate frequencies because the urine of not all infants was screened for amino acidopathies. The G6PD deficiency frequency is accurate, and the study will be published in detail elsewhere.

TABLE 1

A. Total infants screened for amino acidopathies-11,919

Disorders Identified:
1. Transient tyrosinemia 6
2. Transient cystinuria 16
3. Abnormal blood patterns 5
4. Abnormal urine patterns 3

B. Total number of Negro infants screened for G6PD deficiency-6,366

The number deficient:
Males 379 (11,5%)
Females 77 (2.5%)

The abnormal blood and urine pattern have not been correlated with any clinical disease but appear to be a biochemical individuality. A report of the further study of these patterns will be published at a later date.

In addition to the above deficiencies, other inborn errors of metabolism were detected in Negro children admitted to Freedmen's Hospital for diagnosis and treatment. These include classical galactosemia, branched-chain keto aciduria or maple syrup urine disease, and mucopolysaccharidoses. The galactosemic patient was diagnosed clinically and emzymatically and sucessfully treated. The child with branched-chain ketoaciduria appears to be new variant of this disease and will soon be the subject of a report.

Discussion

Any screening is a compromise between what should be done and the resources available. We have used the methods, especially for the detection of amino acidopathies, which will give us the most information with the least technical effort. Althought the Efron method limits us somewhat with sensitivity and the resolution for certain individual amino acids, we believe that observing the patterns of all possible blood amino acids is advantageous. A recent study of 36,000 neonates has indicated that hyperaminoacidemia was found in 316 of these.[12] All but 6 were transient. Six or 1 : 6000 were not transient (with 5 hyperphenylalaninemias and 1 case of hypermethioninemia). This gives some indication of the frequency of hyperamino acidemias in the general population. Since there has not been any complete study published on specific hyperamino acidemias in the Negro, there is more reason to screen for all possible amino acids in this population.

Many states still require screening for PKU only in the newborn population, but 1 study has indicated that the frequency in the general population of homocystinuria has at least approached that of PKU, or about 1:12,000.[13] It is for this reason that we screen urine samples from newborns when available and all followup urine specimens for cystine and homocystine.

Unfortunately, it is not possible to obtain a urine specimen on all newborns before they are discharged because the nurses object to the added burden in their duties and a trial period had indicated to us that simply placing filter paper inside the diaper ended up with fecal contamination. Another problem is with the follow-up specimen (blood and urine on filter paper sent through the mail) which is obtained when a mother takes her infant to the physician for the first time, usually at about 1 month. Although we screen some 99% of the infants born at Freedmen's Hospital for amino acids, our followup percentage is only about 25%. This means that we will miss the late-manifesting diseases that are not prevalent prior to the hospital discharge (2-3 days after birth). Other screening programs around this country have ranged up to 86% for individual amino acids.[14] We intend to increase our follow-up percentage by an active education program involving the mothers while they are confined to Freedmen's Hospital after the delivery of their infants.

In addition, we realize that the screening of blood specimens without the corresponding urine will give only half the picture of the infant's amino-acid levels since several conditions exhibit a normal blood amino-acid level with an increased urine excretion due to the low kidney threshhold for the particular molecule. Thus, ideally, blood at birth, and both urine and blood should be screened sometime between 2 and 5 weeks. This would allow sufficient time for detection and treatment and hopefully prevention of any permanent brain or other organic damage.

Summary

Some 11,919 Negro infants born at Freedman's Hospital have been screened for amino acidopathies and other inborn errors of metabolism.

The results indicate that several types of transient amino-acid imbalances were prevalent in this population while no permanent varieties were found on screening. Several were picked up in hospitalized patients. These included 1 case of galactosemia, 1 case of branched-chain keto aciduria (maple syrup disease), and 3 cases of mucopolysaccharidoses. Of 6,368 newborns screened, 11.5% of all males and 2.5% of all females were found to be G6PD deficient. These were confirmed by a quantitative assay. The indications for screening and some problems encountered have been discussed.

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Pulmonary Hypertension Due to Chronic Upper Airway Obstruction

Report of a Case*

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Although the syndrome of chronic upper airway obstruction, pulmonary hypertension, and congestive heart failure is a well defined, clinical entity among pediatric cardiologists,[1-10] its recognition by the practicing pediatrician may be lacking. In fact, it is given only passing attention in the latest edition of a popular pediatrics textbook. Since the practicing pediatrician is the one who sees most of the children who may have upper airway problems, e.g., enlarged tonsils and adenoids, he should be made more aware of this entity.

It is the purpose of this report to describe a patient with the syndrome of pulmonary hypertension secondary to chronic upper airway-obstruction in order to re-emphasize its importance. Also, in the light of the present tendency in pediatrics to avoid unnecessary removal of tonsils and adenoids, this report reminds us that this syndrome is indeed one of the important, urgent indications for tonsillectomy and adenoidectomy.

Report of Case

T.J. (F.H. No. 323848), a 2½-year-old black male, was admitted to Howard University's Freedmen's Hospital on April 9, 1971 because of respiratory difficulty, chronic mouth breathing, and noisy respiration of 1 year's duration. Actually at 1 year of age he was referred to the otolaryngology clinic of another hospital because of tonsillitis and difficulty in breathing. Since that time he had been followed as an outpatient at the hospital. His mother reported that stertorous respirations and mouth breathing began toward the end of his first year of life. The respiratory distress became much worse at night when he was in bed. This resulted in marked difficulty with both inspiration and expiration, especially the latter, and was accompanied by grunting and subcostal and intercostal retractions. Periodic breathing was also noted. It was at these times that cyanosis was frequently observed by his parents. Several times during each night he had to sit up in bed to breathe properly. This meant that he had not obtained a full night of restful sleep for approximately 1 year prior to this admission. During the episodes of marked breathing difficulty he appeared very somnolent, practically semicomatose, and his eyes rolled back. Although such episodes occurred daily for over the previous 12 to 15 months, his parents had adjusted to these occurrences and pointed out that they became frightened only when cyanosis occurred. They felt that if anything was seriously wrong, it would have been brought to their attention during his numerous clinic visits. They did note, however, that his face had become rather puffy during the previous 6 months and that he appeared lethargic most of the time.

Because of a recent upper respiratory infection a chest X-ray film was taken which revealed cardiomegaly. He was then seen by the pediatric cardiologists who noted the severe respiratory distress and also auscultatory signs of pulmonary hypertension. He was then transferred to our hospital for further evaluation and relief of his airway obstruction.

The child was the product of a full-term pregnancy, complicated by kidney infection and hypertension. He weighed 11 lbs, 9 oz at birth. Except for croup at 3 months of age and pneumonia and tonsillitis at 1 year, his first year of life was unremarkable. Three siblings are being treated for skin allergies.

Physical examination on admission revealed a normally developed 2½-year-old child (weight - 13.4kg, height - 88cm) having marked respiratory distress with stertorous respirations and extreme subcostal, supraclavicular, and intercostal retractions. The lips were slightly cyanosed. He was quite somnolent and difficult to arouse. There was generalized puffiness of the face, and he exhibited an adenoid facies with typical mouth-breathing. A mucopurulent nasal discharge was present, and the eyes kept rolling back. Marked tonsillar hypertrophy was present such that the tonsils met in the midline, but they were free of exudates. The neck veins were slightly distended. On auscultation of the chest, very poor air exchange was noted bilaterally. Examination of the heart revealed a right ventricular lift along the left sternal border. The first heart sound was normal, while the second sound was split with a loud snapping pulmonary component. A grade 2/6 short ejection systolic murmur was noted at the 2nd and 3rd left intercostal spaces. No diastolic murmurs were present. The liver was 2cm below the right costal margin. The remainder of the examination was not remarkable.

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Laboratory data included hemoglobin of 14.0 gm %, hematocrit of 42%, and blood-gas studies as shown in table I which revealed respiratory acidosis. The chest X-ray film (fig. 1) showed generalized cardiomegaly, and lateral neck films (fig. 2) revealed enlarged adenoids impinging on the nasopharyngeal space. The electrocardiogram taken on admission revealed P pulmonale and right ventricular hypertrophy (fig. 3).

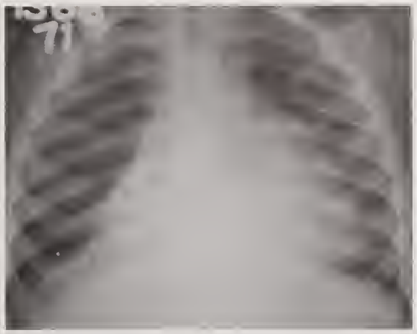

FIG. 1. Chest film before removal of tonsils and adenoids. There was persistence of slight cardiomegaly 6 months postoperative.

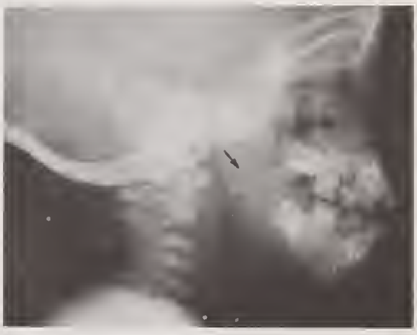

FIG. 2. Lateral neck film showing enlarged soft tissue mass (arrow) encroaching on and almost obliterating the nasopharyngeal space.

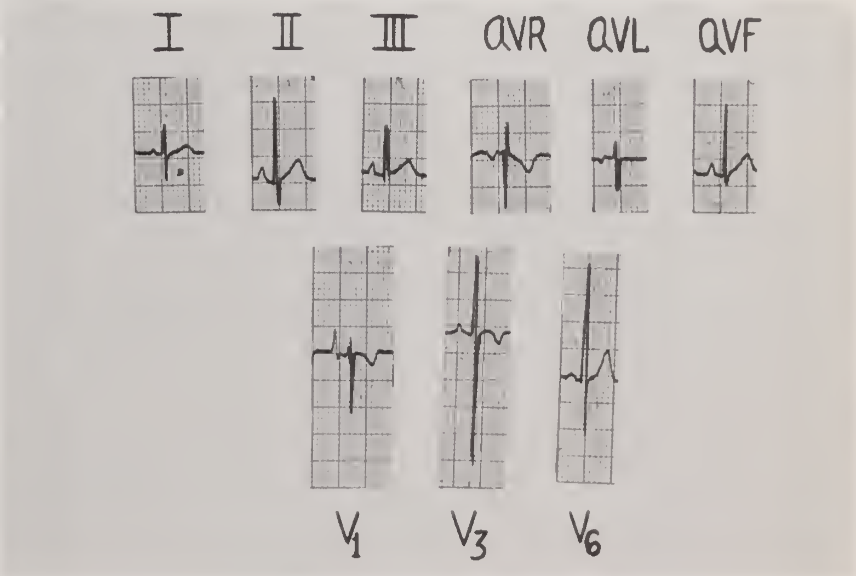

FIG. 3. Electrocardiogram before removal of tonsils and adenoids. Note very tall spiked P-waves in lead V_1, and evidence of right ventricular hypertrophy (large S-wave in V_6).

TABLE I: Blood Gases

Time	pH	pCO_2 (mmHg)	pO_2 (mmHg)
On admission	7.45	54	60
6 hours after admission (Asleep, in O_2 cyanotosis, difficult breathing)	7.28	82	20

Because of the severity of the respiratory distress, lack of improvement with oxygen administration, and the blood-gas changes, tracheostomy was performed by the otolaryngologist less than 24 hours after admission. This resulted in immediate relief of the respiratory distress. Following this, the blood gases returned to normal.

Seventy-two hours after admission, cardiac catheterization was performed, and the results of this study are summarized in table 2.

Following the cardiac catheterization an adenoidectomy and tonsillectomy were done. The tracheostomy tube was removed a few days later. The patient showed no signs of respiratory difficulty following removal of the tracheostomy tube. After dismissal from the hospital he did extremely well with good activity, no breathing problems, and normal sleep at night.

Six months after removal of the adenoids and tonsils the electrocardio-

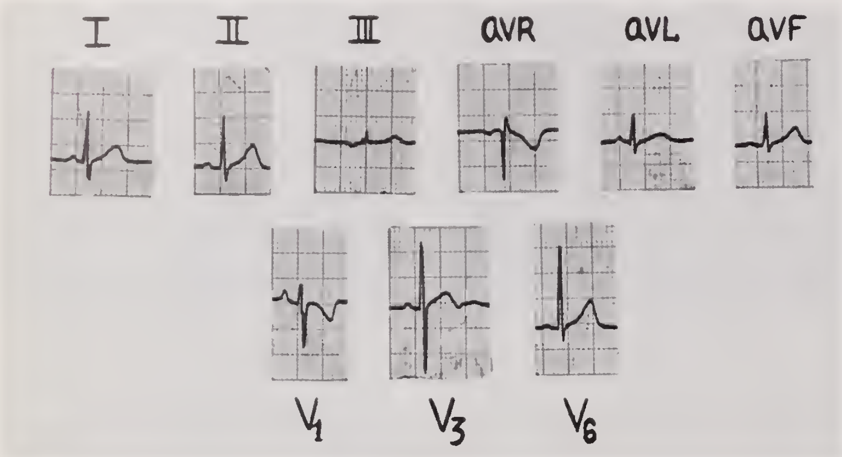

FIG. 4. Normal electrocardiogram 6 months after removal of tonsils and adenoids.

gram had returned to normal (fig. 4), but the chest film still revealed minimal cardiomegaly.

Discussion

This patient demonstrates the development of heart disease due to chronic upper airway-obstruction. Since 1965 when Noonan[1] reported on 2 children who developed cor pulmonale due to hypertrophied tonsils and adenoids, several case reports have appeared in the literature.[3-12] The clinical picture in all of these reports is strikingly similar. There is a history of recurrent upper respiratory-tract infection, noisy breathing, especially while asleep, lethargy, signs of CO$_2$ narcosis when placed in high oxygen environment, cardiomegaly, auscultatory signs of pulmonary hypertension, the development of right-heart failure, and the onset of major symptoms between the ages of 2 and 6 years. Some of these reports have shown cardiac catheterization data[1, 3-11] which demonstrate pulmonary hypertension and wide variations in pulmonary-artery pressures with changes in breathing patterns. Our case serves as a reminder that while this syndrome is not common, it represents an important clinical problem with a relatively simple solution.

This child started at about 1 year of age to have stertorous breathing, and when he was only 2½ years of age when seen by us, he could be categorized as having chronic upper airway-obstruction. This is important since it appears to take time to develop the clinical picture described above.

TABLE 2: Cardiac Catheterization Data*

Site	Pressure (mmHg)	O$_2$ Content (Vol. %)	O$_2$ Saturation (%)
RA	10/0 (6)	12.2	65.0
RV	35/5	12.8	68.0
MPA	35/15 (25)	12.8	68.0
RPA	35/15 (25)	12.8	68.0
Desc. aorta	95/50 (70)	17.5	93.0

Cardiac Index–3.28 L/min./m^2
Pulmonary vascular resistance–480.8 dyne sec. cm-5/M^2

* Obtained 72 hours after the patient was tracheotomized. RA = right atrium, RV = right ventricle, MPA = main pulmonary artery, RPA = right pulmonary artery. ( ) = mean pressure.

The etiology of this clinical syndrome is fairly well understood. It has been shown that hypoventilation from upper airway-obstruction leads to retention of CO$_2$. This results in a respiratory acidosis with a concomitant decrease in the blood pH. Hypoventilation also results in a decrease in the arterial pO$_2$. The combined effects of hypoxia, hypercapnea and acidosis cause an increase in the pulmonary vascular resistance. This increase results from a reactive vasospasm of the pulmonary artery and leads to an increase in the pulmonary-artery pressure. The increase in pulmonary arterial resistance and pressure is reflected by increase in right ventricular and right atrial pressures. Ultimately the right heart may fail, resulting in hepatomegaly and peripheral edema. These pathophysiologic phenomena are summarized in figure 4.

That hypoventilation can lead to cardiac enlargement and heart failure has been known for some time as indicated by Cayler's[13] report in 1961 on the cardiorespiratory changes of the Pickwickian syndrome. The blood-gas changes occurring in a variety of hypoventilation syndromes have been studied. Vogel and Blount[14] and Simmons[15] showed that an increase in hydrogen-ion concentration led to an increase in pulmonary-artery pressure. Rudolph and Yuan[16] later showed in animal studies that when the blood pH is lowered significantly into the acidotic range, the pulmonary vasculature became more reactive to hypoxia and thus higher pulmonary pressures could be obtained.

The blood gases in our patient indicated significant hypercapnea and a rapidly developing acidosis during periods of increased hypoventilation as when he was asleep. Lying in bed in the supine position also probably resulted in more mechanical obstruction from the enlarged tonsils and adenoids.

With chronic hypoventilation, the respiratory center in the brain becomes less responsive to CO_2 and may depend entirely on hypoxia for stimulation. For this reason, these patients become worse when placed in a high oxygen environment since their hypoxia stimulus to respiration is diminished or abolished and more hypoventilation results. Indeed, the blood gases of our patient 6 hours after admission were taken with the patient asleep in the oxygen tent. Since he did not improve, a tracheostomy was done to relieve his extreme difficulty in breathing. If possible, then, a high oxygen environment should be avoided in these patients.

We did not perform cardiac catheterization prior to the tracheostomy. A review of cases from the literature, however, revealed that some patients still had moderate elevations in their pulmonary-artery pressures 6 to 8 months after removal of the airway obstruction. In others it returned to normal and remained so after the obstruction was removed. In our patient we found slight elevation of pulmonary-artery and right-ventricular pressures even though previous tracheostomy had completely bypassed the airway obstruction. Following removal of the tonsils and adenoids all of the symptoms disappeared.

The electrocardiogram which showed signs of right-atrial enlargement and right-ventricular hypertrophy had reverted to normal 6 months after removal of the obstructing tonsils and adenoids. At that time, also, auscultatory findings revealed that the pulmonary component of the second heart sound was now of normal intensity, suggesting that the pulmonary and right ventricular pressures had returned to normal. The heart on the X-ray film, however, appeared to show persistent, slight cardiomegaly. The exact reason is not known, but perhaps Vogel's[14] theory of hyperreactors to hypoxia and hypercapnea can give a possible explanation. Bland[10] has suggested a hypothalamic defect which results in continued mild hypoventilation even though the upper airway obstruction has been removed.

From a clinical standpoint all of these patients can obtain remarkable relief of their cardiorespiratory difficulty by removal of the tonsils and adenoids. This syndrome represents one of the true indications for tonsillectomy and adenoidectomy and points to the need to look for the possible cardiovascular effects in any child with evidence of chronic upper airway-obstruction.

Summary

The case of a 2½-year-old black male child with pulmonary hypertension secondary to hypertrophied tonsils and adenoids has been presented, including the results of right-heart catheterization and blood gas changes.

The patient serves as a reminder of this uncommon clinical problem in that he had almost all of the reported findings except heart failure. Caution in the use of oxygen in these patients was emphasized. Removal of the tonsils and adenoids resulted in complete reversal of all symptoms and reversion of the electrocardiogram to normal after a 6-month follow-up period. The syndrome of pulmonary hypertension resulting from hypertrophied tonsils and adenoids is one of the true indications for tonsillectomy and adenoidectomy.

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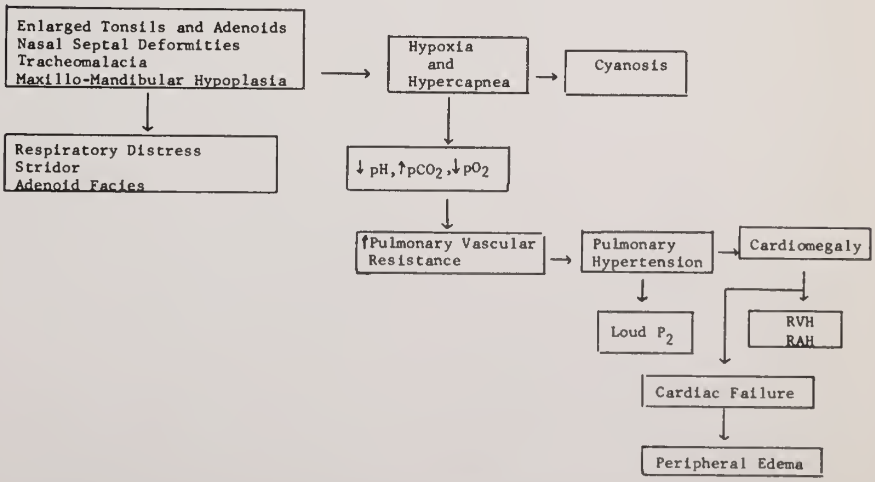

FIG. 5. Schema of events in pulmonary hypertension due to chronic upper respiratory airway-obstruction.

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Comparison of the Cancer Deaths in the Black and White U.S.A. Population from 1949 to 1967

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This study was undertaken to determine the present and future needs of the black U.S. population for cancer care. Begun in July 1970, it has been supported by the Metropolitan Washington Regional Medical Program Grant RPG-1533 (Cancer Radiotherapy Unit) and since June 1, 1971 by the National Cancer Institute Grant ME-RPG 1619 (Howard University Cancer Center Exploration Study).

Twenty years ago the age-adjusted cancer mortality rates were about 10% lower for the black than for the white U.S.A. population.[1] It was widely believed that this was due to underreporting of cancer in Blacks, and that the cancer mortality rates in Blacks and Whites were essentially similar with the exception of cancer of the skin (higher in Whites) and cancer of the cervix (higher in Blacks).

In recent years several epidemiological studies have shown that a number of other cancers are significantly more frequent in U.S. Blacks than in U.S. Whites. Seidman[6] (1970) found this in a New York City study covering the years 1949 to 1951 for cancer of the esophagus, stomach, liver and biliary passages, pancreas, prostate, cervix and bladder. Krain[5] (1970) reported that the mortality from pancreas cancer is greater for U.S. Blacks than for U.S. Whites, and that it is among the highest in the world. Wynder, Mabuchi and Whitmore[8] (1971) came to the same conclusion for cancer of the prostate.

To our knowledge, however, no comprehensive comparison of the cancer mortality in the black and white U.S. population has been made. This is surprising, since the basic data are readily available from the "Official Vital Statistics of the U.S.A."[1] Even a casual appraisal of the black and white cancer deaths in these volumes shows that Black cancer deaths are rising much faster than White cancer deaths. This trend is clearly apparent already from the data up to 1960 but apparently has failed to attract attention.

Method

The data presented in this paper are based entirely on the "Official Vital Statistics of the U.S.A." which are published yearly in 2 large volumes. The last year for which these volumes are available is 1967. For the beginning of this study we chose the year 1949, because a revision of the "International Classification of Diseases" was accepted in 1948. Our study covers, therefore, the most recent 19-year period.

For all graphs we used the year 1949 for both races as the reference point and calculated the changes in subsequent years in percentage of the cancer deaths in 1949. The resulting increases or decreases were entered in the graphs.

With the help of our IBM-2741 terminal linked to an IBM-360/50 computer of the Howard University Computer Center, we then determined the straight line $y = a+b\,(x-49)$, which best fitted the values. In this formula, "y" represents the % increase; and "x" represents the year. The slope "b" and the "y"-intercept "a" was found by means of the method of least squares. The computer also gave the standard deviations of each of these estimates. In figures 1-3 the computer-calculated straight lines are shown. In table 1 the slope of these lines, called the average yearly increase, is given in % together with its standard deviation. If the differences between the average yearly change for Blacks and Whites are greater than 2.04 times the "average standard deviation" ($\sqrt{s_1^2 + s_1^2}$), it is significant at the 95% confidence level; and if it is greater than 2.75 times the average standard deviation, it is significant at the 99% confidence level.

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All Cancers

All Cancers in Both Sexes. Figure 1 shows the increase of the deaths from all cancers in both sexes. Black U.S. cancer death rose from 15,419 to 29,715, an increase of 93%; White U.S. cancer deaths rose from 190,286 to 279,502, and increase of 47%. The computer-calculated yearly increase was 4.98% ± 0.11% in Blacks, and 2.45% ± 0.10% in Whites. Thus, cancer deaths in U.S. Blacks increased on the average of 2.03 times faster than in U.S. Whites. This difference is statistically significant at the 99% confidence level ($p < 0.01$).

All Male Cancers. Figure 2 shows the increase of all male cancer deaths. Black male cancer deaths rose from 7,168 to 16,590, an increase of 131%, while white male cancer deaths rose from 96,450 to 151,541, an increase of 57%. The computer-calculated yearly increase was 7.08% ± 0.18% in black males, and 3.06 ± 0.09% in white males. Thus, male cancer deaths increased in U.S. Blacks 2.31 times faster than in U.S. Whites ($p < 0.01$).

All Female Cancers. Figure 3 shows the increase of all female cancer deaths. Black female cancer deaths rose from 8,251 to 13,125, an increased of 59%, while white female cancer deaths rose from 93,827 to 128,051, an increase of 37%. The computer-calculated yearly increase was 3.16% ± 0.08% in black females, and 1.86% ± 0.08% for white females. Thus, the female cancer deaths increased in U.S. Blacks 1.70 times faster than in U.S. Whites ($p < 0.01$).

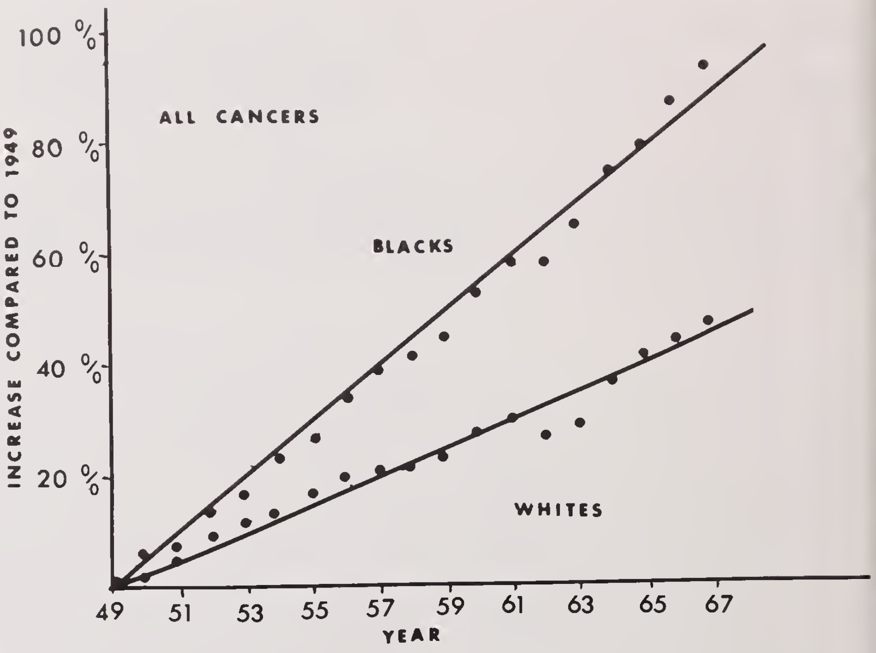

FIG. 1. Increase of Cancer Deaths in Both Sexes U.S.A. 1949-1967.

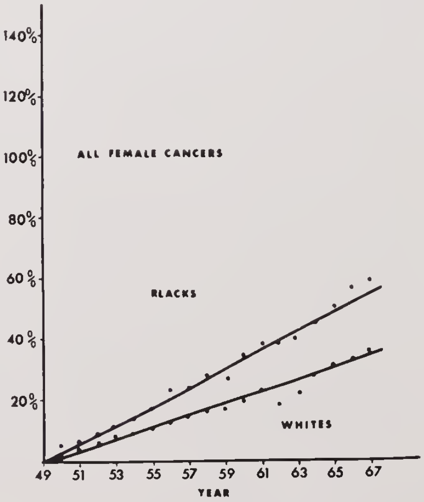

FIG. 2. Increase of Male Cancer Deaths U.S.A. 1949-1967.

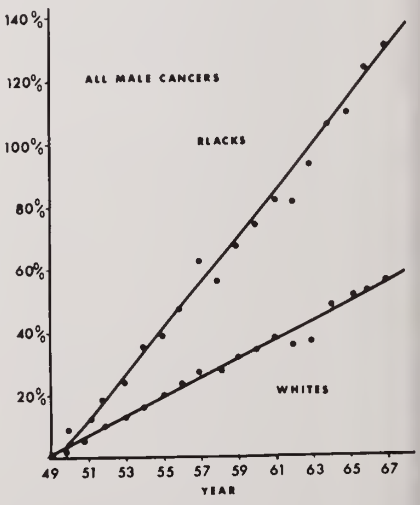

FIG. 3. Increase of Female Cancer Deaths U.S.A. 1949-1967.

Major Individual Cancers

Similar curves as shown in figures 1-3 were prepared for all cancers which caused more than 1,000 cancer deaths in 1967. Space does not permit the reproduction of these curves. The computer-calculated average yearly increases between 1949 and 1967 are given in table 1, where the 24 major U.S. cancers are arranged in decreasing order of the number of deaths they caused in 1967.

The table shows that the largest average yearly increase in U.S. Blacks has occurred in cancer of the lung (21.79%) followed by connective tissue cancer (12.55%), pancreas cancer (11.30%), lymphosarcoma combined with reticulum-cell sarcoma (8,53%), brain cancer (8.46%), esophagus cancer (8.38%), kidney cancer (8.38%), larynx cancer (8.37%), pharynx cancer (7.20%), prostate cancer (7.18%), leukemia (7.17%), ovary cancer (6.21%), colon cancer (6.01%), tongue cancer (5.80%), bladder cancer (5.46%), liver cancer (5.35%), and breast cancer 4.91%). For all these cancers the number of deaths in Blacks has more than doubled since 1949. In Whites only lung cancer (11.51%), connective-tissue cancer (11.36%), and lymphosarcoma combined with reticulum-cell sarcoma (7.75%) have more than doubled from 1949 to 1967.

Stomach cancer, cervix cancer, and rectum cancer, which have decreased in Whites, remained essentially unchanged in Blacks. Lymphosarcoma combined with reticulum-cell sarcoma, Hodgkin's disease, and connective-tissue cancer showed no statistically significant differences in the trend of black and white U.S. cancer deaths since 1949. All other differences are statistically significant at the 99% confidence level, except brain cancer, in which the difference was significant only at the 95% confidence level.

Age-Adjusted Cancer Mortality Rates

While the total number of cancer deaths is the key figure for the planning of clinical facilities for the treatment of cancer patients, the so-called "age-adjusted cancer mortality rates" are the key figures for epidemiological considerations.

TABLE 1: Average Yearly Increase of Deaths from the Twenty-four Most Frequent Cancers–USA: 1949-1967

Cancer Type (1967 deaths)	% U.S. Blacks	% U.S. Whites
1. Lung (54,407)	21.79 ± .89	11.51 ± 0.29
2. Colon (33,082)	6.01 ± 0.28	2.28 ± 0.10
3. Breast (28,217)	4.91 ± 0.17	2.57 ± 0.12
4. Stomach (17,050)	0.08 ± 0.11	−1.94 ± 0.05*
5. Pancreas (16,886)	11.30 ± 0.30	4.92 ± 0.09
6. Prostate (16,345)	7.18 ± 0.22	2.06 ± 0.14
7. Leukemia (14,336)	7.17 ± 0.35	3.75 ± 0.17
8. Rectum (10,431)	1.08 ± 0.27	−0.08 ± 0 12*
9. Ovary (9,168)	6.21 ± 0.26	3.44 ± 0.14
10. Bladder (8,762)	5.46 ± 0.34	1.67 ± 0.10
11. Lympho sarcoma and reticulum-cell sarcoma (7,788)	8.53 ± 0.50	7.75 ± 0.17
12. Cervix (7,411)	0.57 ± 0.20	−0.66 ± 0.15*
13. Brain (7.153)	8.46 ± 0.58	4.17 ± 1.89
14. Kidney (5,894)	6.06 ± 0.35	3.55 ± 0.11
15. Esophagus (5,627)	8.38 ± 0.39	1.67 ± 0 13
16. Skin (4,724)	3.66 ± 0.29	2.10 ± 0.14
17. Biliary tract (4,593)	3.60 ± 0.70	0.62 ± 0.34
18. Hodgkin's (3,446)	2.37 ± 0.36	1.99 ± 0.15
19. Pharynx (2,910)	7.20 ± 0.54	2.47 ± 0.16
20. Larynx (2,797)	8.37 ± 0.46	2.25 ± 0.13
21. Liver (2,152)	5.35 ± 0.91	1.75 ± 0.53
22. Bone (1,853)	−0.03 ± 0.38*	−1.23 ± 0.15*
23. Tongue (1,544)	5.80 ± 0.66	1.72 ± 0.19
24. Connective tissue (1,374)	12.55 ± 1.05	11.36 ± 1.26

* Minus (−) indicates a decrease.

Age-adjusting is necessary because the black population increased faster from 1949 to 1967 than the white population (from 15,549,000 to 23,942,000 versus 134,942,000 to 179,920,000). Another reason is that a greater percentage of Blacks are in younger age-groups in which cancer is not as frequent. In 1967, for instance, 55.2% of all U.S. Blacks were under 25 years old, while only 45.4% of all U.S. Whites were under 25 years of age.

As the base year for our age adjustment we chose the year 1960, because it is the census year closest to the middle of the period under study. The use of the 1960 data results in slightly higher cancer mortality rates than if the age adjustment is made for the year 1940 (as is done in the "Official Vital Statistics of the U.S.A."), because the percentage of people in the most cancer-prone age-groups over 50 years of age was about 3% larger in 1960 (23.3%) than in 1940 (20.4%).

Unfortunately, for the age-adjustment calculation we have available only the population figures for Whites and non-Whites, and thus we cannot give direct figures of the black and white age-adjusted cancer mortality rates.

Of the 24,087,000 non-Whites in the U.S.A. in 1967, 21,984,000 (91.3%) were listed as Blacks. The remaining 2,103,000 (8.7%) are listed in the U.S. statistics as Indians, Chinese, Japanese and "Others."[1] Relatively few cancers are recorded for the latter group; its crude cancer mortality rate of 86 per 100,000 is much lower than for Blacks who had a crude cancer-mortality rate of 138 per 100,000. The age-adjusted cancer mortality rates for Blacks are therefore probably a little higher than for non-Whites.

Table 2 shows the mortality rates (as usually given per 100,000 population) for all cancers, age-adjusted to the 1960 total U.S. population. The rates for both sexes combined show that in 1949 the mortality rate was 8% lower for non-Whites (137.5 per 100,000) than for Whites (149.1 per 100,000). By 1967 the situation was reversed, and the non-White cancer mortality rate (181.5 per 100,000) was 18% higher than the White cancer mortality rate (154.0 per 100,000). The change from 1949 to 1967 was +32% for the non-Whites, while it was only +3% for Whites.

This trend is more pronounced for males, in whom the increase from 1949 to 1967 was 61% for non-White males and 14% for White males. For females, the increase for non-White females from 1949 to 1967 was 6%, while for White females, a decrease of 9% took place during the same period. Age-adjusted mortality rates for the major individual cancers, as well as graphs, will be presented in a forthcoming paper in *Cancer*.

TABLE 2: Mortality Rates Per 100,000 Population For All Cancers, Age Adjusted To 1960 U.S. Population

Sex	Race	1949	1967	Per Cent Change
Both Sexes	Non-Whites	137.5	181.5	+32
	Whites	149.1	154.0	+ 3
Males	Non-Whites	129.9	208.9	+61
	Whites	149.9	171.5	+14
Females	Non-Whites	145.2	154.0	+ 6
	Whites	148.8	135.4	– 9

Causes of Increase

Without more detailed studies, one can only speculate at this time about the causes for the more rapid rise of cancer deaths in the black U.S. population. It seems obvious that there are a number of different causes, and that they contribute in different degrees to the increase of each individual cancer. The most important factors in our opinion are:

Differences in exposure to carcinogens. For cancer of the lung, larynx and bladder one may suspect that in the last decades the number of black cigarette smokers has increased faster than the number of white cigarette smokers. For cancer of the esophagus, the pharynx, the tongue, the liver, and the pancreas an increasing exposure to alcohol must be considered as a possibility for the faster rise of those cancers in the black population. Other carcinogens in food and in the environment may also contribute significantly to the faster increase of cancer in U.S. Blacks.

Dietary habits. Two of the most important cancers, namely, cancer of the colon and cancer of the stomach, appear to be linked to dietary habits, although it is by no means clear what the essential factors in the diet are. For these cancers it might be quite illuminating to study the different dietary habits in the black and white population, and also to consider the rapid changes which have taken place over the last 2 decades in the diet of U.S. Blacks.

Inferior socioeconomic conditions. Cancer of the cervix has a definite link to inferior socioeconomic conditions. Comparative studies of housing and habits in U.S. Blacks and Whites might give new clues to this and other cancers, such as penis and liver cancer, for which an association with socioeconomic conditions has been shown.

Greater incidence of predisposing diseases. Predisposing diseases should also be considered in looking for the reasons for the differences in cancer deaths in Blacks and Whites. This might be especially fruitful for cancer of the prostate and of the pancreas, for which inflammatory diseases have been suspected as predisposing causes.

Less accurate death certificates. Differences in the accuracy of the death certificates may be a possible cause of under-reporting of some cancers in U.S. Blacks. For difficult-to-diagnose cancers like cancer of the pancreas, this might well be an important factor and might make the steep rise of pancreas cancer in U.S. Blacks over the last 20 years more pronounced than it is.

Errors in the census enumeration. Omissions in the enumeration of the census have been estimated at 3.0% and erroneous counting of persons more than once at 1.3%, which indicates a net under-enumeration of 1.7%. This figure is probably higher for non-Whites and may be as high as 7% for non-White single young males (Spiegelman 1968).[7] This would tend to increase the cancer mortality rate in Blacks.

Lower cancer cure rate. For cancers with a very low cure rate, such as cancer of the pancreas or cancer of the lung, differences between Black and White cure rates would make little difference for the number of cancer deaths. However, for cancers for which the cure rate is around 50%, such as cancer of the colon, cancer of the breast, and cancer of the cervix, differences in the cure rates might influence the number of deaths significantly. For us there is little doubt that more Black cancer patients come in advanced stages for treatment than White patients, and that the results are correspondingly poorer in Blacks than in Whites.

Genetic differences. Genetic differences in the susceptibility to cancer must also be considered in looking for the causes of the more rapid rise of the Black cancer deaths. This is difficult to establish for cancers in which exposure to carcinogens is a major factor. However, for cancer of the brain, lymphosarcoma combined with reticulum-cell sarcoma, Hodgkin's disease, bone and connective tissue cancers, for which no external causes have been demonstrated, true genetic differences in the susceptibility could more easily be investigated. According to the studies done at Howard University by Barber and associates,[3] brain cancer is less frequent in Blacks. The same might also be the case for malignant lymphomas, eye, and testes cancers. For connective-tissue sarcoma, the reverse might be true: Crawford et al[4] concluded, on the basis of our own tumor registry, that cancer of the connective tissue is more frequent in Blacks. It is well known that benign tumors of the connective tissue in form of keloids are much more frequent in Blacks.

Implications

It appears tragic that the Black U.S. population, already burdened with so many handicaps, is experiencing a rise in cancer deaths which is twice as fast as in the White U.S. population. The magnitude of the problem may be assessed by calculating the difference between the actual Black cancer deaths and the Black cancer deaths which would have occurred if the increase in cancer deaths had been the same as in U.S. Whites. Instead of 29,715 Black cancer deaths in 1967, we would then have only 22,666 Black cancer deaths in 1967, or 7,049 fewer Black cancer deaths less per year.

To appreciate the significance of this steadily widening cancer mortality gap between the Black and White U.S. population, one must consider the emotional and economic implications. Being stricken by cancer is dreaded in the U.S.A. more than anything else. A poll mentioned by Baker (1971) showed that 62% of the U.S. population listed cancer as the fate they are most afraid of. Blindness was a distant second, with 18% in this poll, and heart disease was third with 9%.[2] The economic burden posed by a cancer patient in the family also is unusually high. Few Black families have the resources which are required for optimal cancer care. Thus, a reduction of the excess Black cancer deaths would be a major contribution, and it appears likely that a relatively minor investment in this field would pay great practical dividends.

By throwing light on the etiology as well as on the value of various preventive measures for cancer detection and cancer treatment in U.S. Blacks, such a study would also give important clues to optimal cancer management in all other races.

Summary

From 1949 until 1967 the number of deaths from all cancers increased twice as rapidly in the Black as in the White U.S. population (5.0% vs. 2.5% per year). The increase was much higher for Black males (7.1% vs. 3.1% per year) than for Black females (3.21% vs. 1.9% per year). About 7,000 fewer cancer deaths would have occurred in 1967 in U.S. Blacks if the increase in cancer deaths since 1949 had been the same as in U.S. Whites.

Of the 24 most frequent U.S. cancers, 18 recorded a more rapid increase of the cancer deaths in Blacks than in Whites from 1949 to 1967. Three cancers, namely, stomach cancer, cervix cancer, and rectum cancer, remained essentially unchanged in Blacks, while they decreased significantly in Whites. Only lymphosarcoma combined with reticulum-cell sarcoma, Hodgkin's disease, and connective-tissue cancer showed no statistically significant differences in Blacks and Whites.

The age-adjusted mortality rates for all cancers in both sexes were 8% lower in 1949 in Blacks than in Whites. By 1947 the situation was reversed and Blacks had an 18% higher cancer mortality rate than Whites. This trend to higher cancer mortality rates is especially pronounced in black males.

Factors which may be a cause for the greater increase of the cancer deaths in U.S. Blacks are higher exposure to carcinogens, dietary habits, inferior socioeconomic conditions, greater incidence of predisposing diseases, less accurate death certificates, errors in the census enumeration, lower cure rates, and genetic differences in the susceptibility to cancer. The relative importance of these factors is different for each individual cancer.

A more detailed analysis of the magnitude and of the causes of this cancer gap is essential to find ways to stem the rapid rise of cancer deaths in the black U.S. population. Such a study would also give clues to the etiology, prevention and treatment of many cancers in all races.

Postscript

After completion of this study we obtained the recently published Monograph 33 of the National Cancer

Institute, "Patterns in Cancer Mortality in the United States 1950-1967." This report by Dr. Fred Burbank is an outstanding example of computerized evaluation of cancer mortality data. It confirms largely our conclusions on the alarming rise of the Black cancer mortality, and we will discuss it in detail in our forthcoming paper in *Cancer*. In the comments to this report, as well as in a paper published on the basis of this report in the New England Journal of Medicine, August 19, 1971 p. 461-462, Burbank stresses the sexual differences, as is apparent from the title of his paper, "Males Dominate Once Again: U.S. Cancer Mortality." The racial differences are mentioned only casually in the last sentences (rate of increase of cancer per year per 100,000:4.1 for black males vs. 1.2 for white males, and 0.03 for black females vs. a decrease of −0.92 deaths per 100,000 for white females), and no comment is offered.

Acknowledgement

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Graduate Educational Profile of a Predominantly Black Medical Class

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In the past there has been a general belief that Black medical school graduates tend to pursue careers in general practice rather than specialty practice in medicine.[1-3] A review of the literature shows that there have been few specific studies designed to answer this question. With the development of Black awareness and newer opportunities available for Black physicians in the United States, we thought it would be of interest to survey the graduating class of 1970 from the Howard University College of Medicine in terms of their plans for continued training in the various medical specialties.

Method of Survey

A questionnaire was prepared and mailed to the hospitals where the graduates were performing their internships. If there was no response to the initial questionnaire, a second was mailed. This report is based on a total of 62 final returns from a class of 93 graduates.

Data Collected

The essential data collected consist of 1) the hospital selected for internship, 2) plans for continued formal medical training after internship, and 3) first and second choices for residency training.

Internship

The distribution of hospitals chosen for internships with respect to type and location are as follows:

Type of Hospitals	No. of Graduates	Per Cent of Respondents
University	32	51.6
Community	20	32.2
U.S. Public Health Service	4	6.4
Army, Navy, Veterans	6	9.6

Location of Hospital	No. of Graduates	Per Cent of Respondents
East	20	32.2
West	10	16.1
North	10	16.1
South	21	34.0
Other (Honolulu)	1	1.6

State locations of hospitals chosen were California, District of Columbia, Hawaii, Illinois, Maryland, Massachusetts, Michigan, New Jersey, New York, Ohio, Pennsylvania, Texas, and Wisconsin.

Plans for Residencies

The 62 questionnaires returned from 66% of the total graduates indicated that 61 physicians planned to pursue residency training. Specialities listed as first choice and second choice are shown in tables 1 and 2 respectively.

Specialties Selected by Women

Eighteen % of the 62 graduates were women. Their choice of residencies in the various specialties are pediatrics 45%, internal medicine 27%, orthopedics 9%, pathology 9%, and child psychiatry 9%.

Discussion

This study was designed mainly to secure a profile of the choice of medical specialties by members of the graduating class of 1970. Tables 1 and

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TABLE 1: Specialties Listed as First Choice

Choice of Specialty	Number of Graduates	Per Cent of Total
Anesthesia	1	1.6
Family Practice	3	4.8
Internal Medicine	10	16.1
Obstetrics/Gynecology	10	16.1
Ophthalmology	4	6.4
Orthopedics	4	6.4
Pathology	1	1.6
Pediatrics	9	14.5
Psychiatry	4	6.4
Radiology	5	8.0
General Surgery	7	11.2
Urology	2	3.2
Not Decided	1	1.6
None	1	1.6
Total	62	100

TABLE 2: Specialties Listed as Second Choice

Specialty	Number of Graduates	Per Cent of Total
Anesthesia	1	1.6
Dermatology	2	3.2
Family Practice	7	11.22
Internal Medicine	4	6.4
Neurology	1	1.6
Obstetrics/Gynecology	2	3.2
Ophthalmology	1	1.6
Pathology	2	3.2
Pediatrics	2	3.2
Psychiatry	2	3.2
Radiology	6	9.3
General Surgery	7	11.2
Urology	1	1.6
None	24	38.9
Total	62	100

2 show the % distribution and number of physicians at the internship level planning to pursue specialty training. It can be seen from these data that, of 62 physicians responding 1 was undecided and 1 had definitely decided not to pursue residency training. Thus 95% of those engaged in internship training planned to continue their medical training in the form of hospital residencies.

Further analysis of the data shows that the highest priority for chosen specialties was essentially evenly divided among internal medicine, obstetrics and gynecology, pediatrics, and general surgery. However, a wide spectrum of specialty areas were chosen by these graduates.

Summary

This survey based upon a questionnaire presented to members of the 1970 graduating class of the Howard University College of Medicine during their internship reveals that:

1. About 50% of the respondents selected University hospitals and about 30% selected community hospitals for internships.
2. Hospitals selected for internships were situated mainly in Eastern and Southern states.
3. About 95% of those actively participating in internship training planned to continue their formal medical training in the form of hospital residency programs.
4. Highest priority for specialty training was fairly evenly divided among internal medicine, obstetrics-gynecology, pediatrics, and surgery.
5. Among the women respondents 45% selected pediatrics as their first choice for specialty training.

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Treatment of Middle Class Heroin Abusers: Preliminary Observations*

DAVID H. FRAM, M.D.† AND HOWARD A. HOFFMAN, M.D.‡

Heroin use has been endemic among disadvantaged, poor, black, inner-city people for many years. More recently, heroin use among these groups has become epidemic. In order to treat this epidemic, most of the major cities of the United States, including Washington, D. C., have been developing government-sponsored treatment programs which have included methadone administration and other rehabilitative services.[1] The private sector of medicine, in contrast, has remained virtually dormant in interest and capacity to develop treatment facilities.

During the past four or five years another change in the patterns of drug abuse of even greater proportions has appeared. Drug usage among white, middle-class youths in suburban areas, which had previously been minimal, has markedly increased. A significant number of these youths have been using heroin and other narcotic drugs.

Although the social and psychological causes of drug abuse among middle-class suburban groups has been the subject of much discussion, insufficient attention has been devoted to the development of appropriate techniques and facilities for treatment. Traditional psychotherapy in outpatient settings for heroin users has not been an effective tool. Methadone maintenance for long-term addicts who are motivated to discontinue heroin use has been shown to be successful,[2] but for short-term heroin users, who are ambivalent about giving up heroin, it is a procedure that raises questions both of ethics and efficacy. Methadone programs, moreover, must be administered with proper checks and controls in order to prevent abuse of the methadone.[3] Inpatient facilities have not shown their effectiveness in treating the addict and enabling him to live in the outside world as a nonaddict without continued treatment.

It seemed to us that a multimodality facility, which could provide a wide variety of therapeutic approaches appropriate to the situation would be an important model to explore, and that such a facility, which would be providing various psychotherapeutic approaches, as well as administering medication, would be best centered in a comprehensive mental-health center.

Developing such an approach from the private sector offers many advantages, including freedom of staff selection, program flexibility, intimacy of contact, and confidentiality. Until recently the attention of private physicians has been directed primarily to the treatment of overdose and detoxification. The rehabilitation aspect of treatment has been seen either as a sociologic and criminal problem or an impossible psychotherapeutic endeavor. It is our view that the total treatment of addiction is indeed a medical responsibility and that models for this treatment need to be developed. Because of these factors and the encouragement of the Medical Society of the District of Columbia, the Psychiatric Institute of Washington developed a Drug Abuse Rehabilitation Center, PIDARC.

Description of Program

Since July of 1971 we have been operating PIDARC at 2101 K St., N. W., Washington, D. C. The facility is located on the second floor of an office building, apart from but close to other sections of the Psychiatric Institute. It is currently staffed by three part-time psychiatrists, a registered nurse, a vocational counselor, an ex-addict mental-health counselor, and an administrative secretary.

Upon admission to the program all patients receive a complete psychiatric, medical, and laboratory examination plus a vocational diagnostic evaluation. Upon completion of these, a diagnostic conference is held and a treatment plan is devised. Patients are accepted into the program if they indicate they have some motivation to discontinue drug use and to work on changing maladaptive behavior patterns. Since we expect patients and their families to be ambivalent about changing ingrained patterns of behavior, our policy is to engage ourselves in this struggle and to discharge a patient only if he does not respond. Each patient is assigned to a team psychiatrist and ex-addict mental-health counselor who work together on the treatment of a group of fifteen patients. All patients attend weekly group-therapy sessions and family meetings, which are co-led by the psychiatrist and mental-health counselor. In smaller groups and individually, all patients meet additionally with the mental-health counselor. All patients have vocational counseling and are given assistance in formulating educational and vocational plans with specific assistance in finding placement. In addition to being available to patients during the open hours of the Center, staff are on call 24 hours a day for emergencies.

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Methadone is available when indicated for detoxification or for maintenance. All methadone is prescribed by the psychiatrist and is dispensed by the nurse. In Phase I of the treatment all methadone dispensed is given in liquid form and is consumed in front of the nurse. In Phase II, which may occur from 60 to 90 days after the beginning, methadone may be dispensed for home consumption one or two days a week if the patient has been involved in therapy and is considered trustworthy. In Phase III, which may occur after six months, the patients may take a six-day supply of methadone home. All patients and staff on the program have regular urine testing for drugs. All patients on Phases I and II have urine testing at least twice weekly.

Coordinated with the outpatient PIDARC program described above is an inpatient drug-abuse treatment-service in the Psychiatric Institute for those patients who require hospitalization. The 2 services operate in close relationship so that a smooth transition from inpatient to outpatient treatment can be effected.

Observations and Comment

After four months of operation we have been able to make a number of observations and preliminary conclusions. Over this period we have had 49 applicants for the program. Of these, 35 have been accepted for treatment. Some of those who did not get into treatment were unable to because they could not pay the fee. Several declined because they were not able to commit themselves to treatment. Of those who were admitted to the program, 31 were white and four black. Nearly all of the patients who were admitted to the program had intact families, with whom the patient either was living or had a close involvement with, and nearly all have at least a high-school education. The average age was 24 years, ranging from 16 to 58 years. Nearly all of the patients have had experience with taking heroin for at least a year. Most had been using heroin from 1 to 4 years. About one-third of the patients being treated are on an abstinence program, about one-third are on detoxification with methadone leading to abstinence, and about one-third are on methadone maintenance. Of the 35 patients who have been admitted to the program since it started, 13 have terminated. One of those was discharged by request of the staff because it was clear that he had continued to use drugs and was not involved in the therapy. The other patients who dropped out of their own accord were extremely ambivalent about their treatment throughout their involvement with us and apparently made the decision that they were not going to change their life styles. This conclusion is reinforced by a comparison of the results of the urine testing between patients who remain with the program and those who terminated it. Those still in the program have had 22 positive urines out of 294, or 7.6 per cent. Those who terminated had 22 positive urines out of 102, or 22 per cent. One of the patients still in treatment accounted for 10 of the 22 positive urines in this group. This patient gave up heroin only after very intensive work. If the data from this patient are excluded, the percentage of positive urines for the group in treatment is 4.5.

These percentages are considerably lower than the 33 per cent positive urines commonly reported in large city programs. We attribute this to a number of factors. Our program is very involving and demanding of the patients. Positive urine tests are quickly brought into the therapy discussions and the reasons for the deviant behavior discussed. Those who do not want to change rapidly sense they are in the wrong place and leave. Also, our patients differ from those in the usual program in that they are mostly white, middle class, suburban, better educated, and motivated by their families. Such factors may be important variables affecting the course of treatment. Since many of our patients have had previous failures in treatment elsewhere, we exclude the explanation that our patients are healthier and less dependent on drugs.

Of the 35 patients admitted to the program, 11 have required hospitalization during some part of their treatment. There appear to be a number of situations which made hospitalization necessary. The patient's drug use may be greatly out of control. Detoxification from mixed addictions that included barbiturates and from addiction to excessive doses of methadone are examples. Another reason for hospitalization has been the existence of a psychosis along with the addiction. Another has been for added control for a patient beginning an abstinence program when his heroin craving is severe. In some situations the family becomes so unable to function when the addict's condition is first discovered that the beginning of treatment has to occur with the addict hospitalized. When the family's anxiety is lessened, treatment can then be shifted to an outpatient basis.

We have made other observations of the patients who have come to this treatment center. Most of them have had readily identifiable psychiatric difficulties which could be conceptualized in psychiatric terms, and these concepts used in the conduct of therapy. Many are passive-dependent, apathetic people who are having serious identity problems. Very few have had problems which were mostly related to their taking heroin and which could be treated primarily with methadone. Some have described the heroin experience as not only extremely pleasurable but one that also enabled them to be apathetic with less discomfort. In many of the patients psychological dependence on narcotics appears to be as profound a problem as physiologic addiction. Rather than a fear of withdrawal symptoms, it is the overwhelming impulse to have the pleasure of a heroin experience that results in repeated episodes of drug usage. These people, frequently young, middle-class and suburban, have not yet experienced the social disintegration and degradation of leading the "street life," as it has been described for inner-city addicts.

Our criteria for methadone maintenance reflect these observations. Patients are considered for methadone maintenance if they are at least 18 years old, if their addiction history is at least a year, and if they have previously failed in a serious treatment attempt while abstinent. Maintenance appears to be an effective treatment for the patient who is disenchanted with the life of the heroin addict but is unable to overcome the heroin craving that interferes so greatly with any therapeutic work. Methadone treatment does not appear to be effective for those patients still looking primarily to get 'high'.

The weekly multiple family groups have become a focal point in the treatment program at PIDARC. All too often the relationships in the family have deteriorated to the point where the addict is regarded by the family with suspicion, fear and hostility. The family has devoted much effort in attempting to regulate the addict's life in order to prevent drug use, and interacts little in other ways. We have found that when we take on a major role in the management of the patient's addiction and provide a setting for the family members to talk, they are freed to explore other aspects of family life. Communication among family members has been facilitated, leading both to the exposing of, and resolution of family conflicts. Family members have felt relieved of some of their burden and also have felt the support and understanding of group members from other families. There have been some situations in which the family pathology was sufficiently great to recommend more intensive family therapy.

Conclusion

We have described a private, multimodality facility for the treatment of patients with severe drug-abuse problems. This facility was developed as a private sector model for treating a problem usually left to governmental or non-medical concern. Society is just awakening to the idea that addiction is a medical problem, and the medical profession needs to involve itself in its solution. PIDARC is an example of such an involvement by the private sector.

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Sickle Cell Anemia, a Public Health Problem in the District of Columbia

RAYMOND L. STANDARD, M.D., M.P.H.*

Sickle Cell disease is a major public health problem of young Blacks. In the District of Columbia, where 70 per cent of the population is Black, this inheritable disease is particularly significant. Table 1 illustrates the population trend in the District of Columbia.

TABLE I: Negro Population Trend District of Columbia, 1950-70

Year	Total Population	% Negro
1950	802,200	35
1954	798,800	42
1960	763,956	56
1965	802,000	63
1967	793,500	67
1968	NA	NA
1969	NA	NA
1970	755,500	70

The prevalence of Sickle Cell Anemia is greater among the District's children than other health problems such as diabetes, nephrosis, cystic fibrosis and phenylketonuria (PKU). Table 2 provides a comparison of the estimated prevalence of Sickle Cell Anemia and other diseases in four major U.S. cities.

In the D.C. Department of Human Resources' Community Health Services' clinics, children are tested when Sickle Cell Anemia is suspected during diagnostic workup for episodic illnesses. These tests are also given to women during their first pregnancy and to infants in high risk categories. However, the Department has not been able to conduct a massive program to screen and diagnose this blood disorder.

Recently, sickledex tests were performed on 708 of the blood samples drawn from children for lead level determinations. Table 3 illustrates that 59 or 8.3 per cent of the 708 samples were positive for Sickle Cell trait. Positive screening tests were confirmed by electrophoresis.

This percentage is in keeping with figures quoted from the literature for Blacks who live in the United States; the incidence of Sickle Cell trait ranges from 7 per cent to 13.4 per cent. The average figure most frequently quoted is 8.5 per cent.

There is a need for greater public and professional awareness plus financial support for Sickle Cell Anemia screening, testing, counseling and treatment. Since the majority of Blacks in the United States now live in urban areas, Sickle Cell Anemia has become a public health concern for those persons involved in urban health planning.

A comprehensive program of health education, early detection, and counseling of those with Sickle Cell disease can reduce the incidence of this disorder. Prompt treatment and long term follow-up can reduce morbidity and early mortality.

TABLE II*: Prevalence Estimates: Selected Long-Term Illnesses (White and Negro Cases/100,000 Children)

Diagnosis	Philadelphia, 32% Negro	Richmond, 44% Negro	Detroit, 47% Negro	District of Columbia, 68% Negro
Sickle cell anemia	61	84	89	129
Diabetes mellitus	50	47	46	39
Nephrosis	15	16	16	16
Cystic fibrosis	10	8	8	5

* Roland Scott, "Health Care Priority and Sickle Cell Anemia," J.A.M.A., Vol. 214, No. 4 (October 26, 1970)

TABLE III: Sickle Cell Screening Using Sickledex

Specimen	No. Positive (S. Trait)	Percent Positive	Positive by Electrophoresis
708	59	8.3	58*

* Positive specimen (59th) was an abnormal gene not of sickle type.

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* Director of Public Health and Administrator, Community Health and Hospitals Administration, D.C. Department of Human Resources.

In the District of Columbia, there are 379,000 Blacks between one year and 40 years of age. Sickle Cell screening and testing should be concentrated in this age group.

Although it is possible to screen a population of this magnitude, the Department currently does not have the resources nor manpower to carry out such a large program. Therefore, the following program alternatives have been considered:

1. Conduct an extensive educational program to alert the Black Community to the problem, but leave it up to individuals to seek services from existing private and public medical resources.
2. Offer voluntary screening service to all persons who by law must have pre-marital VDRL tests before they can marry.
3. Screen and identify the teenage population and provide counseling prior to marriage.
4. Combine a Sickle Cell screening program with other current health testing programs aimed at the population at greatest risk (9 months to 40 years).

The latter alternative is probably the most realistic considering the current budgetary constraints, and it should provide the greatest yield through the years.

Presently in the District of Columbia there are health screening programs for: 1) pre-school children through the neighborhood health centers and maternal and child health centers; 2) school age children through the school health program; 3) young adults through neighborhood health centers, categorical clinics, and 4) at multiphasic screening units.

A comprehensive Sickle Cell program should consist of the following: an educational program to alert the population to Sickle Cell Anemia; screening, using sickledex test kits in order to find those persons who are carriers of the disease; confirming those with positive reactions through electrophoresis and by hemoglobin determinations; clinical evaluation, treatment and counseling for those who have the disease; and counseling and screening for family members of those who have Sickle Cell disease.

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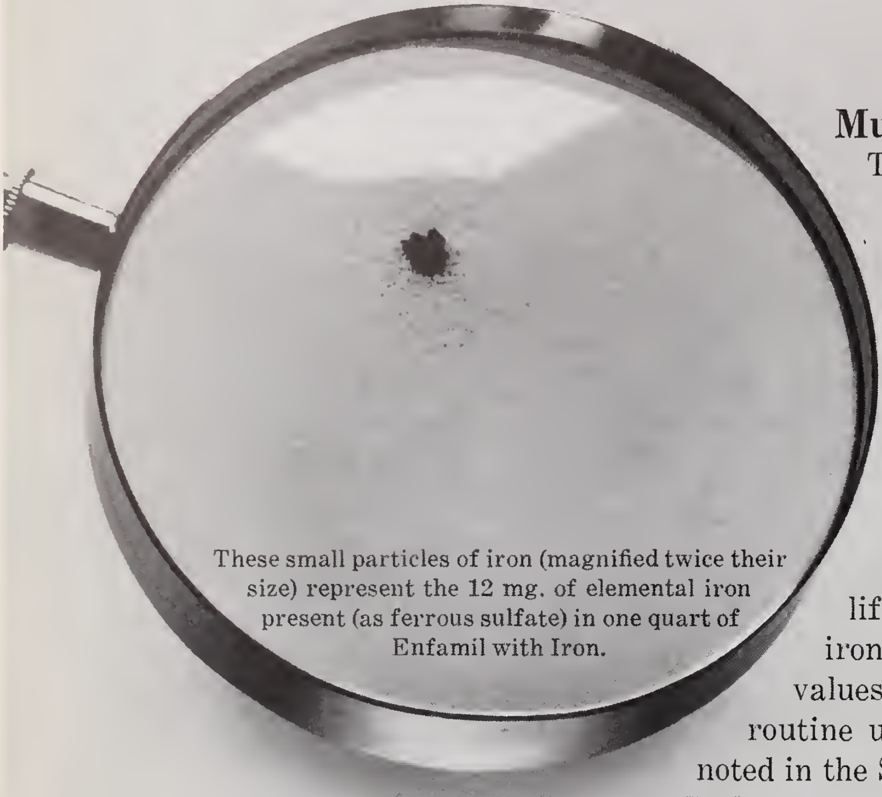

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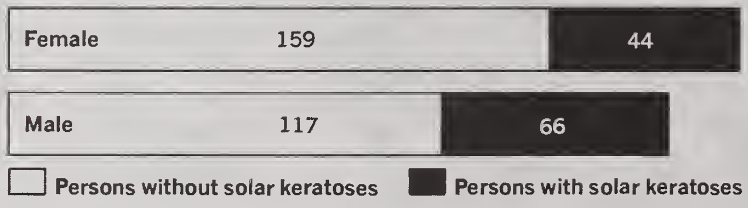
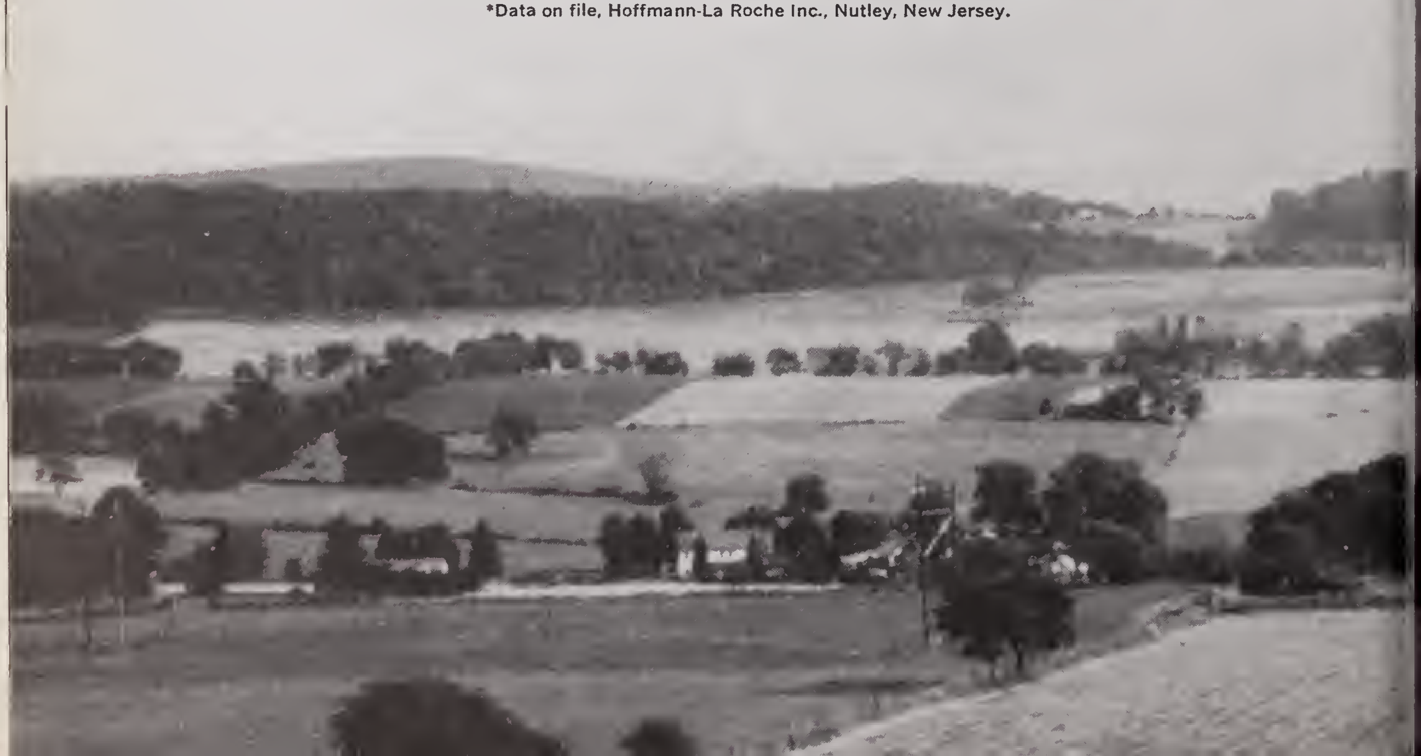
Prevalence of solar keratoses in white persons over 40 in Tipton County, Tennessee



*Data on file, Hoffmann-La Roche Inc., Nutley, New Jersey.



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Adverse Reactions: Local—pain, pruritus, hyperpigmentation and burning at application site most frequent; also dermatitis, scarring, soreness and tenderness. Also reported—insomnia, stomatitis, suppuration, scaling, swelling, irritability, medicinal taste, photosensitivity, lacrimation, leukocytosis, thrombocytopenia, toxic granulation and eosinophilia.
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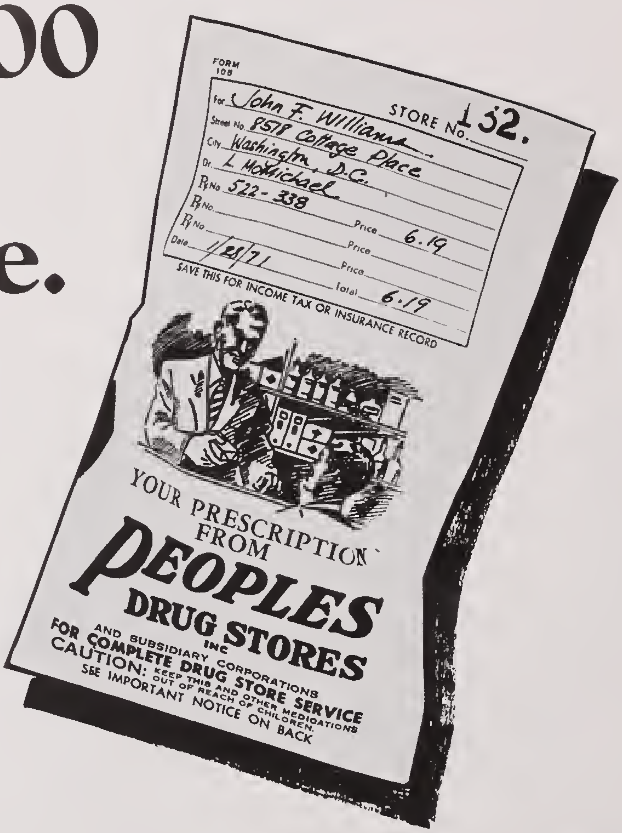

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Minors' Consent to Medical Treatment

The Problem

A 14-year old girl and her 17-year old boyfriend have run away from their homes in California and have been traveling across country. The girl has sustained a bad fall and has severe stomach pains. The pair stumble into your office, clinic or emergency room and ask for help. Legally, what can you do? The answer to this question is different in Maryland, Virginia and the District of Columbia.

An initial problem which the doctor must face when confronted with this situation is how to obtain the girl's consent to any medical examination and/or treatment he determines to be necessary. If the doctor does not first obtain such a consent, he will be subject to potential civil and criminal liability for assault and battery. However, if the doctor can obtain a valid consent this potential liability will be eliminated. The problem for the doctor in our hypothetical situation, of course, is that a minor is deemed incapable under the law of giving a valid consent, so that the 14-year old's parents, apparently residing in California, must be contacted. At best, this task will be difficult, and most certainly will delay the treatment the girl obviously needs.

Many states have realized the difficulty of this situation, and have passed laws to alleviate it. Such states include Maryland and Virginia, where relatively clear standards with respect to minors' consents have been established; unfortunately, this is not the case in the District of Columbia.

District of Columbia

The District of Columbia has no statute relating to the question of how and when a minor's consent to medical treatment is valid. In the absence of such a statute, and because there apparently are no court cases in the District of Columbia concerning this problem, the following general rules of tort law will apply:

The fact that the 14-year old wishes to be treated does not justify the doctor's proceeding with the various tests, examinations and perhaps surgery he deems necessary. He must first obtain the consent of the girl's parent or guardian in order to avoid civil liability and possible criminal sanctions for assault and battery. Let us suppose, however, that telephone calls to California are unproductive; the girl's parents cannot be found. What then?

In most cases, minors who are approaching maturity have been deemed capable of giving a valid consent, at least as to preliminary examinations and minor surgery. Thus, if the girl in our situation were 17 years old, or older, a doctor would probably not be held either civilly or criminally liable for providing treatment short of major surgery for her. If the girl were in need of emergency treatment (*e.g.*, if she were bleeding internally and was in danger of death), various court cases have held that consent requirements would be waived, so that the doctor could perform whatever services (including major surgery) he believed reasonably necessary.

The problem with the above set of "rules", of course, is that they are not rules at all, but merely guidelines. A doctor practicing in the District of Columbia must therefore assess each minor patient's situation as it arises, and then determine if he should proceed, in an attempt to stay within the above "rules."

Fortunately, Maryland and Virginia have enacted laws which set more definite standards under which a doctor can act.

Maryland

In Maryland, the doctor would be free to treat the 14-year old girl in our hypothetical situation without fear of civil or criminal penalties resulting from the minor's lack of capacity to consent. Article 43, Section 135 of the Annotated Code of Maryland (1957) provides that a minor has the same capacity to consent to treatment as an adult in the following situations:

1. The minor is 18 years old or older.
2. The minor is married or the parent of a child.
3. The minor seeks treatment concerning venereal disease, pregnancy, contraception not amounting to sterilization, or drug use.

4. In the judgment of the doctor treating the minor, the delay involved in obtaining parental consent would adversely affect the life or health of such minor.

The statute further provides that no doctor shall be civilly or criminally liable solely on account of the minor's lack of capacity to consent. Thus, provided that *she* consented to treatment our peripatetic girl could be treated by a doctor in Maryland with impunity, even though her situation did not fit one of the categories noted above.

Virginia

The law in Virginia is more stringent than that of Maryland. Section 32-137 of the Code of Virginia (1950) confers authority to consent to treatment of minors *who are separated from the custody of their parents* upon the following persons:

1. If the child is in the custody of a court, Board of Welfare or a state institution, authority to consent is conferred on a judge, Commissioner of Public Welfare or the executive officer of such state institution, respectively.
2. Any child 18 years or older not in the custody of the persons noted in (1) above may also give a valid consent.

The statute also provides that a child may give a valid consent for treatment concerning venereal disease and related diseases, or drug use, birth control, pregnancy or family planning.

There is a catch-all provision in the Virginia statute which states that whenever the obtaining of parental consent is not possible because the parent is not a resident of Virginia, cannot be found, or cannot be consulted with reasonable promptness, then the doctor must obtain the consent of a judge of a juvenile or domestic relations court. Unlike the law in Maryland, the Virginia law does not confer on the minor himself the capacity to consent to treatment except where he is separated from his parents and is 18 or over, or where he needs treatment in connection with venereal disease, birth control and the like. Thus, if the young lady of our hypothetical situation has arrived in Virginia, the doctor treating her would, if he wished to assume no risks in connection with a lack of consent, be obligated to get the consent of a judge of juvenile court. At best, this is a cumbersome procedure, perhaps often ignored despite the attendant risks.

Exceeding Scope of Consent

Even assuming a valid consent under either of the above-noted statutes or the more general rules applicable in the District of Columbia, the doctor must take care not to exceed the scope of the consent given to him. A patient who consents to a minor operation on his nose has not authorized a tonsillectomy. If the patient, minor or adult, is under anaesthetic, the mere desirability of an operation does not protect the surgeon from liability for a battery (any nonconsensual touching), even if the doctor has complied with good professional practice. On the other hand, it is generally held that the doctor may proceed if in surgery a critical, unforeseen emergency arises, or if the consent originally given (typically obtained in most hospitals) is broad enough to cover the surgery performed.

Finally, it should be stressed that the scope of this column has been limited to a doctor's potential liability arising from giving treatment without having first obtained a valid consent. A doctor is still liable for exceeding the scope of such consent as noted above, and is also liable for any negligent acts occurring in the course of surgery or other treatment otherwise validly authorized. The threshhold problem of obtaining consent to treatment of minors, however, will continue to be thorny until more jurisdictions adopt laws similar to the liberal statute presently existing in Maryland.

MICHAEL H. LEAHY, J.D.

Edited by Stefan F. Tucker, J. D., of Arent, Fox, Kintner, Plotkin and Kahn, Washington, D.C.

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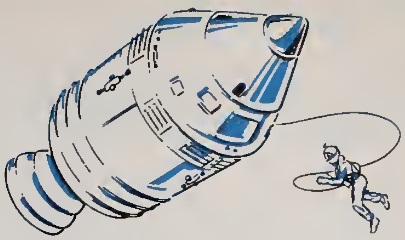

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d.c. MENTAL HEALTH ADMINISTRATION

Forest Haven

An Insistent Gap in Medical Philosophy

Until very recently, children whose level of intellectual functioning enabled them to achieve 80 or above on standard I.Q. tests were taught in the public schools along with other "normals" and low-normals". Children whose I.Q. fell between 60 and 80 were placed in special classes or special schools for the "educable." Children whose I.Q. fell between 40 and 60 were usually sent to some special institution and classified as "trainable". In those same institutions, children were also kept whose I.Q. fell anywhere below 40. It was felt that nothing could be done for these children in the way of education and other than minimal training, so someone was hired to care for them and tend to their basic needs throughout life.

Forest Haven was set up as an institution to care for the trainables, minimally educable, and for those who functioned below trainable levels. It now cares for over 1300 of these.

Under the medical model in which Forest Haven was instituted, adults were hired to care for the basic needs of the children. These were hired at the usual babysitter's pay level, since that was approximately the service asked of them, but they had to be adults in order to physically manage persons entrusted to their care, some of whom would become adult and live at Forest Haven throughout their geriatric years.

No studies have been made concerning the kinds of motivations that enable mature adults to work for years at the lowest pay grades in the Civil Service. One can infer that there is something docile in their makeup, for they soon found themselves acting as houseparents for 30 or more children, teenagers or adults. They are still doing this today.

What does the medical model suggest for the handling and treatment of those with I.Q.'s of 40 or less? Nothing. Keep them clean, fed, and clothed. And presumably out of sight so normal members of their families will not meet with unnecessary stress while trying to achieve and maintain their own life adjustment.

Clothed? Clean? Over 200 teenagers were until recently placed in an old red brick building with bars on every conceivable exit. Clothes were an encumberance so they tore them off continuously. Feces were valued missiles in fights against the staff or with each other, therefore they were stashed away and hoarded whenever possible. Boils and other infections were rampant as a result. The stench became unbearable. Even after scrubbing and cleaning both walls and floors, the odors remained.

They were well fed, with a planned diet that was engineered to keep them in good physical fitness—and at peak energy level. Medical philosophy would not have encouraged clothing that further emphasized their "difference." Thus the usual buttons, zippers, and safety pins in their diapers. And shoes that tied. Normal clothes that, when worn by the mentally subnormal, tend to yield a total picture of gross "difference." At least after an hour or two on a crowded living unit.

And the training given the trainables? Well, those who were trainable presumably learned to tie shoes, button shirts and zip trousers by dent of daily repitition. They learned to feed themselves in a hit and miss kind of way as they were fed by the staff year in and year out. They learned basic toilet skills the same way. In other words they were often trained in the basic living skills in an on-the-job manner as they were guided through them by harried "cottage parents." With any time remaining during the day, an individual child may have received special practice with his buttons or shoe laces under the guidance of the staff. Older children might help in some cases.

Other basic skills such as making his bed, or helping out with the housework were often added as the children showed competence to handle them. Social skills were taught, again on a limited basis, by nonphysical punishments of various kinds, or by small rewards. Thus, a child was taught not to fight with other children, to share his toys at times, to pick up after himself, and to not intrude into private areas of others.

Those who were below the trainable level were allowed to vegetate on hospital wards. They were dressed with minimal clothing—usually diapers that could be changed quickly—they were left to sit for hours in front of a window for entertainment. Or maybe given a rag toy. They were led or wheeled to the dining area for meals, had their clothing changed when necessary, then sat on the floor or a bench again for the remainder of the day.

Many such children had multiple handicaps in addition to their low intellectual level. They could move about somewhat, but could not stand or sit without help. These had to be held by the staff or placed on the floor where they couldn't fall from a height. If placed in a chair, or a bench, or even in bed, they had to be tied loosely so they would not fall when unattended.

At the other end of the subnormal continuum, the educables often were taught basic skills, raising them to a functioning level that allowed their entrance into public classrooms for the exceptional. From there, they might enter a special vocational training program, and later move into the community under minimal supervision, to take their place in society as best they could. They usually were not expected to read at a normal adult level or to do more than basic arithmetic, such as counting change. They were taught how to save their money and basic budgeting skills.

Often, the educables graduated from the special institution to a sheltered workshop and/or a halfway house, where other agencies within Government took over the basic education and training functions while the parent institution provided minimal supervision of the process at best.

Today all of this is changing. Society is no longer willing to see children strapped into a chair or sitting listlessly on a floor in front of a window or television set for hours on end. They will no longer tolerate adolescents and adults striding about unclothed, even for a few seconds. Along with the modern discoveries that even trainables can learn surprisingly complicated skills when taught by behavior modification techniques has come an unwillingness on the public's part to have these children learn less.

The public now demands that these children and adults have active entertainment and recreation instead of passively sitting around while life goes by.

They demand that their living units be clean, well lighted, and sanitary. They demand the latest in medical and psychological diagnosis and treatment. They demand intensive, planned training for the trainable, education for the educable, and rehabilitation for those who are capable of taking their place in normal society. In short, they demand the maximum board-feet of lumber be obtained from this forest; part of America's natural endowment.

Medical philosophy, which is still custodial in nature, is not up to date with these demands. We are moving into the vacuum, however, and in the next issue of the Journal some of the unfolding details of this movement will be outlined.

ESSEX C. NOEL, III, M.D.
Director, D.C. Mental Health Administration

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d.c. HEALTH NOTES

Results of Blood Lead Levels in the Model Cities Area—1971

A special lead poisoning detection program in the Model Cities Area of the District of Columbia has revealed that of the 2,214 children screened, 568 or 25.6 per cent had high levels of lead, i.e., over 40 micrograms per 100 milliliters of whole blood. There were 427 or 19.2 per cent of the children screened with 40 to 49 micrograms of lead in their systems per 100 milliliters of whole blood, while 141 or 6.3 per cent had 50 or above micrograms of lead per 100 milliliters of whole blood.

Thirty children (1.35 per cent of the total screened or 5.3 per cent of the 568 with elevated blood lead levels) were confirmed as suffering from lead poisoning in the Model Cities Area. There was a total of 160 children confirmed as having lead poisoning in all detection programs throughout the District of Columbia.

During the period of the testing—April 1971 through December 1971—children, ages one through six, were tested for lead poisoning in a special mobile unit of the Community Health and Hospitals Administration of the D. C. Department of Human Resources.

The Model Cities Area lead detection program was conducted by the Community Health and Hospitals Administration under a grant of $204,154 from the Model Cities Commission.

The Model Cities Area was divided into 20 wards for the special lead detection project, and it was covered ward by ward by a special lead detection mobile unit. The principal objectives of the lead screening program are early detection and prevention of the mental retardation that is caused by lead poisoning.

In addition to the special Model Cities Project, the Neighborhood Health Centers of the Community Health and Hospitals Administration are open to any parent who wish to have their children tested for lead poisoning. Children seen regularly at selected CHHA well baby clinics are being tested routinely.

A project grant of $527,861 is being requested from the Bureau of Environmental Management by the Department of Human Resources to expand the present program to all areas of the city. The expanded program expects to test 10,000 children, one through

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five years of age, in the 900 Day Care Centers operating in the District. The new Mobile Unit, purchased especially for the Lead Detection Project, will be used in the Day Care Center testing program.

In addition, to the DHR lead screening programs, the Department is encouraging non-government agencies involved in lead screening programs to send their blood samples for analysis to the Central Laboratory of the Department of Human Resources.

Also under the grant, a Central Lead Poisoning Registry is proposed to provide continuity in screening, treatment, and follow-up, and to provide an expanded housing inspection program, utilizing a new mechanical lead detection system.

RAYMOND L. STANDARD, M.D., M.P.H.
Director of Public Health
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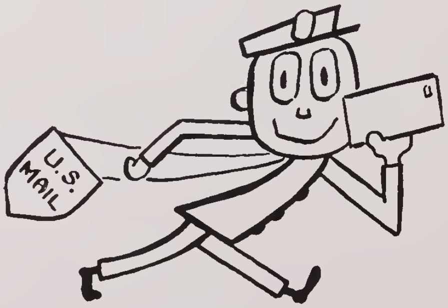

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JAMES EARL HARRELL, M.D. Office: Washington Hospital Center. Born in El Dorado, Arkansas. Medical degree: University of Arkansas, 1962. Specialty: Radiology.

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JOSEPH P. MOCK, M.D. Office: Washington Hospital Center. Born in Bedford, Pennsylvania. Medical degree: University of Pennsylvania, 1963. Specialty: Radiology.

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CEFIE DE LA PAZ, M.F. Office: D.C. General Hospital. Born in the Philippines. Medical degree: Far Eastern University, 1961. Specialty: Anesthesiology.

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Resident-Intern

ASIT P. BASU, M.D. Office: Cafritz Hospital. Born in West Bengal, India. Medical degree: R.G. Kar Medical College, 1959. Specialty: General Surgery.

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BERNARDO KOTELANSKI, M.D. Office: Veterans Administration Hospital. Born in Buenos Aires, Argentina. Medical degree: University of Buenos Aires, 1964. Specialty: Internal Medicine.

ALAN E. RAINESS, M.D. Office: Walter Reed General Hospital. Born in New York, New York. Medical degree: University of Paris, 1965. Specialty: Psychiatry.

Student

Georgetown University. Freshman: Michael E. Jay.

George Washington University. Freshmen: Jeanna C. Miller, Philip C. Ptacin, Don R. Wiesner. Sophomore: John M. Thompson.

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book REVIEWS

Review of Hemodialysis for Nurses and Dialysis Personnel. C.F. GUTCH AND MARTHA H. STONER. Price, $7.50. Pp. 221. St. Louis: C.V. Mosby Company, 1971.

The major appeal of this book is to the new dialysis nurse and technician who have not received formal training in hemodialysis. In addition, as the title indicates, this book serves as a review for established practitioners of dialysis, with an introduction in variation of equipment and techniques. The information presented reveals the vast experience of the authors, obviously possessing a practical knowledge of the basic principles and problems of dialysis.

The material is presented in an unusual style. Each topic is introduced with a narrative and then questions are posed and answered for more explicit information. A breakdown of topics is covered in the Table of Contents.

Practical aspects of dialysis are covered with varying degrees of intensity. The book begins with the hemodialysis team, identifying the role of each member. The second chapter is detailed in basic chemistry and physiology, presenting difficult although essential matter. The chapters continue with good organization of substance, covering in sequence general information about hemodialysis, proceeding through types of dialyzers, communicating advantages and disadvantages of each. Delivery systems are given similar consideration. The dialyzer assembly chapter does not give detailed instruction (manufacturer's instructions are available and should be adhered to) but provides a useful review of major types. Beneficial facts are reported on accessory equipment. Various clean-up procedures are commented upon but only brief mentioning of maintenance and servicing of equipment. Considerable attention is given to cannulas and arteriovenous fistulas. This material would be particularly helpful in alleviating the stress involved with handling cannulas or performing venapunctures.

The discussion of Heparin administration is complete. Patient monitoring is recounted. Acute dialysis is concise but brief. A more detailed canvassing of chronic dialysis ensues with an excellent review of related problems. Also included are the emotional, psychological and social aspects of dialysis in addition to the difficult aspect of diet and fluid management. Peritoneal dialysis is given proper recognition. The book concludes with the need for home dialysis and record keeping.

Here is a concise, soft cover book of 200 pages, imparting useful and pertinent facts concerning dialysis. Not to be ignored is the ease of reading and the low cost.

KATHLEEN KEADY, R.N.

Drugs and Youth: Proceedings of the Rutgers Symposium on Drug Abuse. J.P. WITTENBORN, PH.D., HENRY BRILL, M.D., JEAN PAUL SMITH, PH.D. AND SARAH WITTENBORN. Price, $22.75. Pp. 485 with illustrations. Springfield, Illinois: Charles C. Thomas, 1969.

This is not so much a "proceedings" as it is a kind of World Almanac of drugs and drug abuse. Like the World Almanac, it is eminently useful for settling arguments—do the Swedes abuse amphetamines more than the Japanese, for example? It presents a wonderfully broad cross-cultural view of youthful drug use around the world, past and present. It contains clinical studies of drug effects, statistical analyses of drug use in delineated populations, and legal exegeses of the drug laws. There are 40 separate articles, and not one of them is sloppy or naive. Unlike the proceedings of many symposia, this book is well-indexed and beautifully printed. By now, of course, much of the statistical material in the book is outdated, but "Drugs and Youth" remains the best single volume on the topic.

MICHAEL J. HALBERSTAM, M.D.

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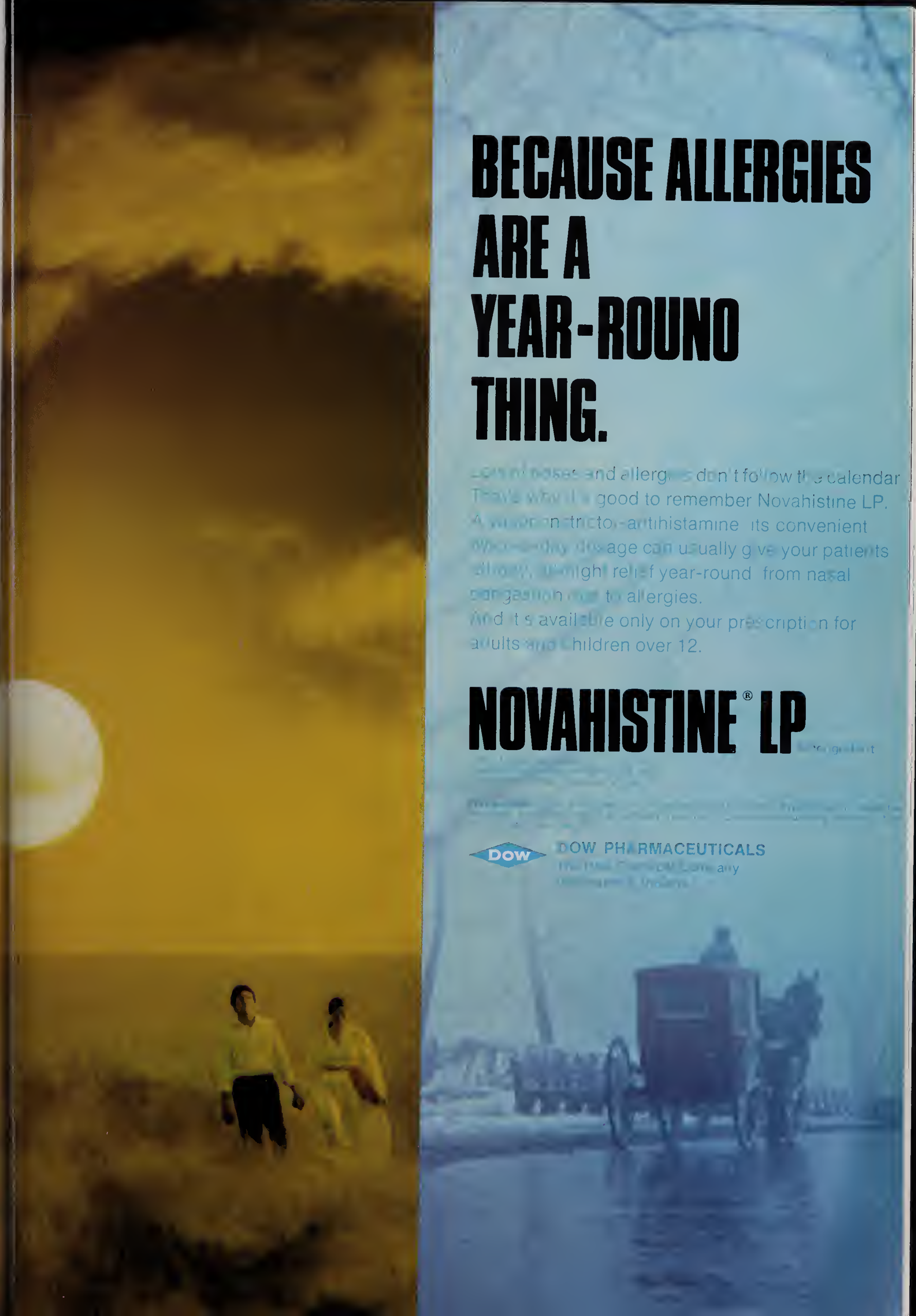


BECAUSE ALLERGIES ARE A YEAR-ROUND THING.

Lots of noses and allergens don't follow the calendar. That's why it's good to remember Novahistine LP. A vasoconstrictor-antihistamine, its convenient twice-a-day dosage can usually give your patients all-day, all-night relief year-round, from nasal congestion due to allergies.
And it's available only on your prescription for adults and children over 12.

NOVAHISTINE® LP Decongestant

[illegible]

DOW PHARMACEUTICALS
The Dow Chemical Company
Indianapolis, Indiana

Femininity & Vaginitis

any women still believe that a uche is a cure-all for vaginal cretions and malodor. Mother ls daughter and the myth is rpetuated.

Other cosmetic products are not ıch better. Though they may be ective in some minor infections, ey cannot touch the real medical oblem, which very often is chomonal vaginitis.

Medicine's most effective cure for trichomonal vaginitis is Flagyl® (metronidazole). It is also pleasantly feminine because it provides the simplicity of oral medication . . . frees women from the unpleasant mess and bother of douches.

When the problem is trichomonal vaginitis . . . remember Flagyl. It cures trichomoniasis with an unmatched high degree of effectiveness.

Flagyl is indicated for the treatment of trichomoniasis in both male and female patients and the sexual partners of patients with a recurrence of the infection provided trichomonads have been demonstrated by wet smear or culture.

Flagyl®

(brand of metronidazole)

Indications: For the treatment of trichomoniasis in both male and female patients and the sexual partners of patients with a recurrence of the infection provided trichomonads have been demonstrated by wet smear or culture. The oral form is indicated also for intestinal amebiasis and amebic liver abscess.

Contraindications: Evidence or history of blood dyscrasia, active organic disease of the CNS, the first trimester of pregnancy and a history of hypersensitivity to metronidazole.

Warnings: Use with discretion during the second and third trimesters of pregnancy and restrict to those pregnant patients not cured by topical measures. Flagyl (metronidazole) is secreted in the breast milk of nursing mothers. It is not known whether this can be injurious to the newborn.

Precautions: Mild leukopenia has been reported during Flagyl use; total and differential leukocyte counts are recommended before and after treatment with the drug, especially if a second course is necessary. Avoid alcoholic beverages during Flagyl therapy because abdominal cramps, vomiting and flushing may occur. Discontinue Flagyl promptly if abnormal neurologic signs occur. Exacerbation of moniliasis may occur. In amebic liver abscess, aspirate pus during metronidazole therapy.

Adverse Reactions: Nausea, headache, anorexia, vomiting, diarrhea, epigastric distress, abdominal cramping, constipation, a metallic, sharp and unpleasant taste, furry or sore tongue, glossitis and stomatitis possibly associated with a sudden overgrowth of *Monilia*, exacerbation of vaginal moniliasis, an occasional reversible moderate leukopenia, dizziness, vertigo, incoordination and ataxia, numbness or paresthesia of an extremity, fleeting joint pains, confusion, irritability, depression, insomnia, mild erythematous eruptions, "weakness," urticaria, flushing, dryness of the mouth, vagina or vulva, pruritus, dysuria, cystitis, a sense of pelvic pressure, dyspareunia, fever, polyuria, incontinence, decrease of libido, nasal congestion, proctitis, pyuria and darkened urine have occurred in patients receiving the drug. Patients receiving Flagyl may experience abdominal distress, nausea, vomiting or headache if alcoholic beverages are consumed. The taste of alcoholic beverages may also be modified. Flattening of the T wave may be seen in EKG tracings.

Dosage and Administration

For Trichomoniasis. In the Female: One 250-mg. tablet orally three times daily for ten days. Courses may be repeated if required in especially stubborn cases; in such patients an interval of four to six weeks between courses and total and differential leukocyte counts before, during, and after treatment are recommended. Vaginal inserts of 500 mg. are available for use, particularly in stubborn cases. *When the vaginal inserts are used, one 500-mg. insert is* placed high in the vaginal vault each day for ten days and the oral dosage is reduced to two 250-mg. tablets daily during the ten-day course of treatment. Do not use the vaginal inserts as the sole form of therapy. *In the Male:* Prescribe Flagyl only when trichomonads are demonstrated in the urogenital tract, one 250-mg. tablet two times daily for ten days. Flagyl should be taken by both partners over the same ten-day period when it is prescribed for the male in conjunction with the treatment of his female partner.

For Amebiasis. Adults: For acute intestinal amebiasis, 750 mg. orally three times daily for 5 to 10 days. For amebic liver abscess, 500 to 750 mg. orally three times daily for 5 to 10 days.

Children: 35 to 50 mg./kg. of body weight/24 hours, divided into three doses, orally for ten days.

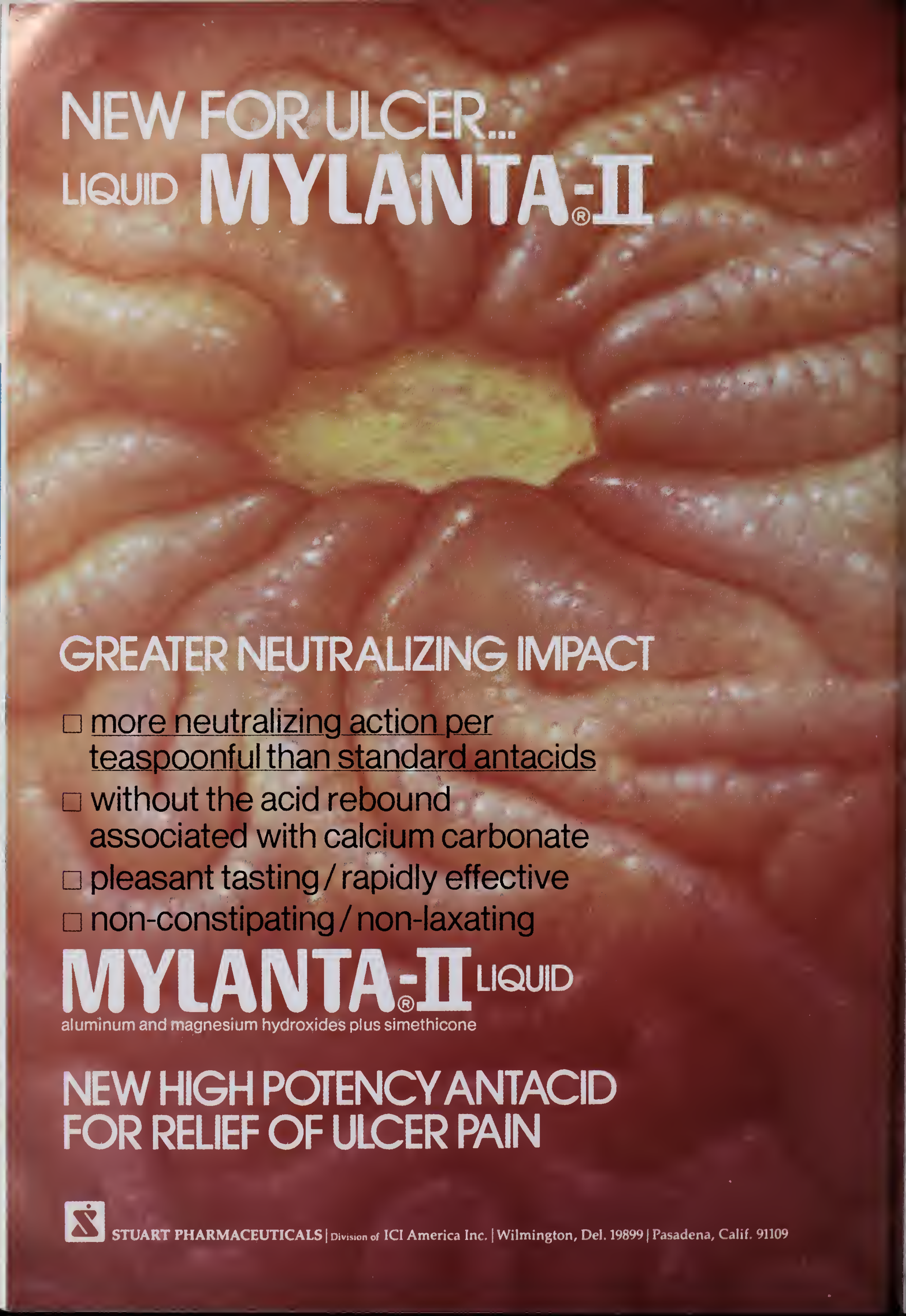
Dosage forms: Oral tablets 250 mg.
Vaginal Inserts 500 mg.

Flagyl® (metronidazole)

SEARLE Manufactured by SEARLE & CO.
San Juan, Puerto Rico 00936

Address medical inquiries to:
G. D. Searle & Co., Medical Department
P. O. Box 5110, Chicago, Illinois 60680

Research in the Service of Medicine



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- ☐ pleasant tasting / rapidly effective
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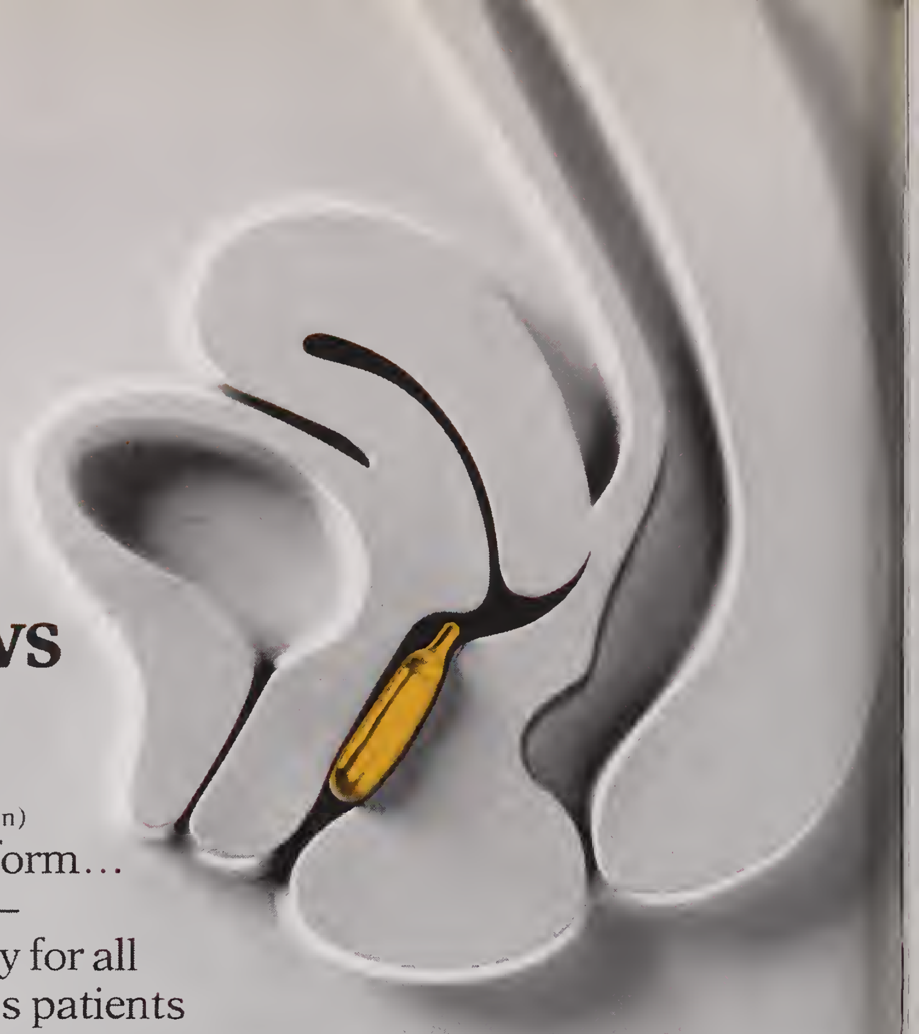
A gratifying announcement about Empirin® Compound with Codeine

CIII

You may now specify up to five refills within six months when you prescribe Empirin Compound with Codeine (unless restricted by state law).

It is significant in this era of increased regulation, that Empirin Compound with Codeine has been placed in a less restrictive category. You may now wish to consider Empirin with Codeine even more frequently for its predictable analgesia in acute or protracted pain of moderate to severe intensity.

Empirin Compound with Codeine No. 3 contains codeine phosphate* (32.4 mg.) gr. ½. No. 4 contains codeine phosphate* (64.8 mg.) gr. 1. *(Warning—may be habit-forming.) Each tablet also contains: aspirin gr. 3½, phenacetin gr. 2½, caffeine gr. ½.



Now form follows function

Only **Candeptin** (candicidin) gives you this unique form... a soft gelatin capsule— highly effective therapy for all your vaginal moniliasis patients

CANDEPTIN® (candicidin) VAGELETTES™ Vaginal Capsules...a unique dosage form... anatomically and therapeutically designed to extend flexibility in the treatment of vaginal moniliasis.

Virtually unlimited application

CANDEPTIN VAGELETTES Vaginal Capsules provide the specific high potency antimonilial agent, candicidin, in a soft gelatin capsule—the shape designed with your patient in mind. It permits easy manual insertion without the need for an applicator or inserter...of particular value for the pregnant patient...for *intravaginal use.* By cutting off the tip of the narrow soft end, the contents can be extruded through an intact hymen for *intravaginal use.* And it is readily adaptable to *topical application* for labial involvement, and/or *intravaginal use* to treat mucosal infection.

CANDEPTIN (candicidin) provides:

Rapid results

Prompt, symptomatic relief—itching, burning, and discharge subside in 48-72 hours.[1]

Soothing, miscible ointment permits complete contact with affected tissue.

Usually cures in a single 14-day course of therapy.[2,3,4]

Safe

Exact dosage assured.[2,3]

No side effects, clinical reports of irritation or sensitization extremely rare.

Convenience

Easy to use intravaginally and/or topically for labial involvement.

Encourages patient acceptance and cooperation.

Therapy is easy to start in your office.

Clinical proof of potency

CANDEPTIN (candicidin) is significantly more potent *in vitro* than nystatin.[5] CANDEPTIN Vaginal Ointment and Tablets have a clinical record of cure rates of 90% and more in pregnant and non-pregnant patients.[1,4,6] In recent studies on CANDEPTIN VAGELETTES Vaginal Capsules, involving both gravid and non-gravid patients, a 100% culture-confirmed cure rate was achieved with a single 14-day course of therapy.[2,3]

Unique

CANDEPTIN® (candicidin)

VAGELETTES™ Vaginal Capsules

Description: CANDEPTIN (candicidin) Vaginal Ointment contains a dispersion of candicidin powder equivalent to 0.6 mg. per gm. or 0.06% Candicidin activity in U.S.P. petrolatum. 3 mg. of Candicidin is contained in 5 gm. of ointment or one applicatorful. CANDEPTIN Vaginal Tablets contain Candicidin powder equivalent to 3 mg. (0.3%) Candicidin activity dispersed in starch, lactose and magnesium stearate. CANDEPTIN VAGELETTES Vaginal Capsules contain 3 mg. of Candicidin activity dispersed in 5 gm. U.S.P. petrolatum.
Action: CANDEPTIN Vaginal Ointment, Vaginal Tablets, and VAGELETTES Vaginal Capsules possess anti-monilial activity.
Indications: Vaginitis due to Candida albicans and other Candida species.
Contraindications: Contraindicated for patients known to be sensitive to any of its components. During pregnancy manual Tablet or VAGELETTES Capsule insertion may be preferred since the use of the ointment applicator or tablet inserter may be contraindicated.
Caution: During treatment it is recommended that the patient refrain from sexual intercourse or the husband wear a condom to avoid re-infection.
Adverse Reaction: Clinical reports of sensitization or temporary irritation with CANDEPTIN Vaginal Ointment, Vaginal Tablets or VAGELETTES Vaginal Capsules have been extremely rare.
Dosage: One vaginal applicatorful of CANDEPTIN Ointment or one Vaginal Tablet or one VAGELETTES Vaginal Capsule is inserted high in the vagina twice a day, in the morning and at bedtime, for 14 days. Treatment may be repeated if symptoms persist or reappear.
Available Dosage Forms: CANDEPTIN Vaginal Ointment is supplied in 75 gm. tubes with applicator (14-day regimen requires 2 tubes). CANDEPTIN Vaginal Tablets are packaged in boxes of 28, in foil with inserter—enough for a full course of treatment. CANDEPTIN VAGELETTES Vaginal Capsules are packaged in boxes of 14 (14-day regimen requires 2 boxes.)

Store under refrigeration to insure full potency.

Federal law prohibits dispensing without prescription.

References: **1.** Olsen, J.R.. Journal-Lancet *85* 287 (July) 1965. **2.** Giorlando, S.W.: Ob/Gyn Dig. *13* 32 (Sept) 1971. **3.** Decker, A.. Case Reports on File, Medical Department, Julius Schmid **4.** Giorlando, S W , Torres, J.F., and Muscillo, G . Am. J Obst. & Gynec. *90*: 370 (Oct. 1) 1964 **5.** Lechevalier, H.: Antibiotics Annual 1959-1960. New York, Antibiotica Inc., 1960 pp. 614-618. **6.** Friedel, H.J. Maryland M J . *15* 36 (Feb.) 1966.

Julius Schmid Pharmaceuticals
423 West 55th Street
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CANDEPTIN® (candicidin)
Vaginal Tablets
Vaginal Ointment
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10
11

rheumatoid arthritic blowup...

Tandearil® Geigy
oxyphenbutazone NF

tablets of 100 mg.

ortant Note: This drug is not a simple analgesic. not administer casually. Carefully evaluate patients ore starting treatment and keep them under close ervision. Obtain a detailed history, and complete sical and laboratory examination (complete mogram, urinalysis, etc.) before prescribing and at uent intervals thereafter. Carefully select patients, iding those responsive to routine measures, con- indicated patients or those who cannot be observed uently. Warn patients not to exceed recommended age. Short-term relief of severe symptoms with smallest possible dosage is the goal of therapy. sage should be taken with meals or a full glass of k. Patients should discontinue the drug and report mediately any sign of: fever, sore throat, oral ions (symptoms of blood dyscrasia); dyspepsia, gastric pain, symptoms of anemia, black or tarry ots or other evidence of intestinal ulceration or morrhage, skin reactions, significant weight gain or ma. A one-week trial period is adequate. Discon- ue in the absence of a favorable response. Restrict tment periods to one week in patients over sixty.
ications: Acute gouty arthritis, rheumatoid arthritis, umatoid spondylitis.
traindications: Children 14 years or less; senile ients; history or symptoms of G.I. inflammation or eration including severe, recurrent or persistent spepsia; history or presence of drug allergy; blood scrasias; renal, hepatic or cardiac dysfunction; pertension; thyroid disease; systemic edema; matitis and salivary gland enlargement due to the g; polymyalgia rheumatica and temporal arteritis; ients receiving other potent chemotherapeutic nts, or long-term anticoagulant therapy.
rnings: Age, weight, dosage, duration of therapy, stence of concomitant diseases, and concurrent ent chemotherapy affect incidence of toxic reac- ns. Carefully instruct and observe the individual ient, especially the aging (forty years and over) o have increased susceptibility to the toxicity of the g. Use lowest effective dosage. Weigh initially predictable benefits against potential risk of severe, n fetal, reactions. The disease condition itself is unaltered by the drug. Use with caution in first trimester of pregnancy and in nursing mothers. Drug may appear in cord blood and breast milk. Serious, even fatal, blood dyscrasias, including aplastic anemia, may occur suddenly despite regular hemograms, and may become manifest days or weeks after cessation of drug. Any significant change in total white count, relative decrease in granulocytes, appearance of immature forms, or fall in hematocrit should signal immediate cessation of therapy and complete hematologic investigation. Unexplained bleeding involving CNS, adrenals, and G.I. tract has occurred. The drug may potentiate action of insulin, sulfonylurea, and sulfonamide-type agents. Carefully observe patients taking these agents. Nontoxic and toxic goiters and myxedema have been reported (the drug reduces iodine uptake by the thyroid). Blurred vision can be a significant toxic symptom worthy of a complete ophthalmological examination. Swelling of ankles or face in patients under sixty may be prevented by reducing dosage. If edema occurs in patients over sixty, discontinue drug.
Precautions: The following should be accomplished at regular intervals: Careful detailed history for disease being treated and detection of earliest signs of adverse reactions; complete physical examination including check of patient's weight; complete weekly (especially for the aging) or an every two week blood check; pertinent laboratory studies. Caution patients about participating in activity requiring alertness and coordination, as driving a car, etc. Cases of leukemia have been reported in patients with a history of short- and long-term therapy. The majority of these patients were over forty. Remember that arthritic-type pains can be the presenting symptom of leukemia.
Adverse Reactions: This is a potent drug; its misuse can lead to serious results. Review detailed information before beginning therapy. Ulcerative esophagitis, acute and reactivated gastric and duodenal ulcer with perforation and hemorrhage, ulceration and perforation of large bowel, occult G.I. bleeding with anemia, gastritis, epigastric pain, hematemesis, dyspepsia, nausea, vomiting and diarrhea, abdominal distention, agranulocytosis, aplastic anemia, hemolytic anemia, anemia due to blood loss including occult G.I. bleeding, thrombocytopenia, pancytopenia, leukemia, leukopenia, bone marrow depression, sodium and chloride retention, water retention and edema, plasma dilution, respiratory alkalosis, metabolic acidosis, fatal and nonfatal hepatitis (cholestasis may or may not be prominent), petechiae, purpura without thrombocytopenia, toxic pruritus, erythema nodosum, erythema multiforme, Stevens-Johnson syndrome, Lyell's syndrome (toxic necrotizing epidermolysis), exfoliative dermatitis, serum sickness, hypersensitivity angiitis (polyarteritis), anaphylactic shock, urticaria, arthralgia, fever, rashes (all allergic reactions require prompt and permanent withdrawal of the drug), proteinuria, hematuria, oliguria, anuria, renal failure with azotemia, glomerulonephritis, acute tubular necrosis, nephrotic syndrome, bilateral renal cortical necrosis, renal stones, ureteral obstruction with uric acid crystals due to uricosuric action of drug, impaired renal function, cardiac decompensation, hypertension, pericarditis, diffuse interstitial myocarditis with muscle necrosis, perivascular granulomata, aggravation of temporal arteritis in patients with polymyalgia rheumatica, optic neuritis, blurred vision, retinal hemorrhage, toxic amblyopia, retinal detachment, hearing loss, hyperglycemia, thyroid hyperplasia, toxic goiter association of hyperthyroidism and hypothyroidism (causal relationship not established), agitation, confusional states, lethargy; CNS reactions associated with overdosage, including convulsions, euphoria, psychosis, depression, headaches, hallucinations, giddiness, vertigo, coma, hyperventilation, insomnia; ulcerative stomatitis, salivary gland enlargement.
(B) 98-146-800-E

For complete details, including dosage, please see full prescribing information.

GEIGY Pharmaceuticals
Division of CIBA-GEIGY Corporation
Ardsley, New York 10502

TA. 8356-9

a new outlook in chronic pain

of moderate to severe intensity

Though Talwin® can be compared to codeine in analgesic efficacy, it is not a narcotic. So patients receiving Talwin for prolonged periods face fewer of the consequences you've come to expect with narcotic analgesics. And that, in the long run, can mean a better outlook for your chronic-pain patient.

Talwin Tablets are:

- **Comparable to codeine in analgesic efficacy:** one 50 mg. Talwin Tablet appears equivalent in analgesic effect to 60 mg. (1 gr.) of codeine. Onset of significant analgesia usually occurs within 15 to 30 minutes. Analgesia is usually maintained for 3 hours or longer.
- **Tolerance not a problem:** tolerance to the analgesic effect of Talwin Tablets has not been reported, and no significant changes in clinical laboratory parameters attributable to the drug have been reported.
- **Dependence rarely a problem:** during three years of wide clinical use, only a few cases of dependence have been reported. In prescribing Talwin for chronic use, the physician should take precautions to avoid increases in dose by the patient and to prevent the use of the drug in anticipation of pain rather than for the relief of pain.
- **Not subject to narcotic controls:** convenient to prescribe—day or night—even by phone.
- **Generally well tolerated by most patients:** infrequently cause decrease in blood pressure or tachycardia; rarely cause respiratory depression or urinary retention; seldom cause diarrhea or constipation. If dizziness, lightheadedness, nausea or vomiting are encountered, these effects tend to be self-limiting and to decrease after the first few doses. (See last page of this advertisement for a complete discussion of adverse reactions and a brief discussion of other Prescribing Information.)

50 mg. Tablets

Talwin®

brand of

pentazocine (as hydrochloride)

the long-range analgesic

a new outlook in chronic pain

of moderate to severe intensity

Contraindications: Talwin, brand of pentazocine (as hydrochloride), should not be administered to patients who are hypersensitive to it.

Warnings: *Head Injury and Increased Intracranial Pressure.* The respiratory depressant effects of Talwin and its potential for elevating cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions, or a preexisting increase in intracranial pressure. Furthermore, Talwin can produce effects which may obscure the clinical course of patients with head injuries. In such patients, Talwin must be used with extreme caution and only if its use is deemed essential.

Usage in Pregnancy. Safe use of Talwin during pregnancy (other than labor) has not been established. Animal reproduction studies have not demonstrated teratogenic or embryotoxic effects. However, Talwin should be administered to pregnant patients (other than labor) only when, in the judgment of the physician, the potential benefits outweigh the possible hazards. Patients receiving Talwin during labor have experienced no adverse effects other than those that occur with commonly used analgesics. Talwin should be used with caution in women delivering premature infants.

Drug Dependence. There have been instances of psychological and physical dependence on parenteral Talwin in patients with a history of drug abuse and, rarely, in patients without such a history. Abrupt discontinuance following the extended use of parenteral Talwin has resulted in withdrawal symptoms. There have been a few reports of dependence and of withdrawal symptoms with orally administered Talwin. Patients with a history of drug dependence should be under close supervision while receiving Talwin orally.

In prescribing Talwin for chronic use, the physician should take precautions to avoid increases in dose by the patient and to prevent the use of the drug in anticipation of pain rather than for the relief of pain.

Acute CNS Manifestations. Patients receiving therapeutic doses of Talwin have experienced, in rare instances, hallucinations (usually visual), disorientation, and confusion which have cleared spontaneously within a period of hours. The mechanism of this reaction is not known. Such patients should be very closely observed and vital signs checked. If the drug is reinstituted it should be done with caution since the acute CNS manifestations may recur.

Usage in Children. Because clinical experience in children under 12 years of age is limited, administration of Talwin in this age group is not recommended.

Ambulatory Patients. Since sedation, dizziness, and occasional euphoria have been noted, ambulatory patients should be warned not to operate machinery, drive cars, or unnecessarily expose themselves to hazards.

Precautions: *Certain Respiratory Conditions.* Although respiratory depression has rarely been reported after oral administration of Talwin, the drug should be administered with caution to patients with respiratory depression from any cause, severe bronchial asthma and other obstructive respiratory conditions, or cyanosis.

Impaired Renal or Hepatic Function. Decreased metabolism of the drug by the liver in extensive liver disease may predispose to accentuation of side effects. Although laboratory tests have not indicated that Talwin causes or increases renal or hepatic impairment, the drug should be administered with caution to patients with such impairment.

Myocardial Infarction. As with all drugs, Talwin should be used with caution in patients with myocardial infarction who have nausea or vomiting.

Biliary Surgery. Until further experience is gained with the effects of Talwin on the sphincter of Oddi, the drug should be used with caution in patients about to undergo surgery of the biliary tract.

Patients Receiving Narcotics. Talwin is a mild narcotic antagonist. Some patients previously receiving narcotics have experienced mild withdrawal symptoms after receiving Talwin.

CNS Effect. Caution should be used when Talwin is administered to patients prone to seizures; seizures have occurred in a few such patients in association with the use of Talwin although no cause and effect relationship has been established.

Adverse Reactions: Reactions reported after oral administration of Talwin include *gastrointestinal:* nausea, vomiting; infrequently constipation; and rarely abdominal distress, anorexia, diarrhea. *CNS effects:* dizziness, lightheadedness, sedation, euphoria, headache; infrequently weakness, disturbed dreams, insomnia, syncope, visual blurring and focusing difficulty, hallucinations (see *Acute CNS Manifestations* under WARNINGS); and rarely tremor, irritability, excitement, tinnitus. *Autonomic:* sweating; infrequently flushing; and rarely chills. *Allergic:* infrequently rash; and rarely urticaria, edema of the face. *Cardiovascular:* infrequently decrease in blood pressure, tachycardia. *Other:* rarely respiratory depression, urinary retention.

Dosage and Administration: *Adults.* The usual initial adult dose is 1 tablet (50 mg.) every three or four hours. This may be increased to 2 tablets (100 mg.) when needed. Total daily dosage should not exceed 600 mg.

When antiinflammatory or antipyretic effects are desired in addition to analgesia, aspirin can be administered concomitantly with Talwin.

Children Under 12 Years of Age. Since clinical experience in children under 12 years of age is limited, administration of Talwin in this age group is not recommended.

Duration of Therapy. Patients with chronic pain who have received Talwin orally for prolonged periods have not experienced withdrawal symptoms even when administration was abruptly discontinued (see WARNINGS). No tolerance to the analgesic effect has been observed. Laboratory tests of blood and urine and of liver and kidney function have revealed no significant abnormalities after prolonged administration of Talwin.

Overdosage: *Manifestations.* Clinical experience with Talwin overdosage has been insufficient to define the signs of this condition.

Treatment. Oxygen, intravenous fluids, vasopressors, and other supportive measures should be employed as indicated. Assisted or controlled ventilation should also be considered. Although nalorphine and levallorphan are *not* effective antidotes for respiratory depression due to overdosage or unusual sensitivity to Talwin, parenteral naloxone (Narcan®, available through Endo Laboratories) is a specific and effective antagonist. If naloxone is not available, parenteral administration of the analeptic, methylphenidate (Ritalin®), may be of value if respiratory depression occurs.

Talwin is not subject to narcotic controls.

How Supplied: Tablets, peach color, scored. Each tablet contains Talwin (brand of pentazocine) as hydrochloride equivalent to 50 mg. base. Bottles of 100.

Winthrop Winthrop Laboratories, New York, N. Y. 10016 (1583)

50 mg. Tablets

Talwin®

brand of
pentazocine (as hydrochloride)

the long-range analgesic

obituaries

Vice Admiral George Wehnes Calver
1887-1972

Retired Vice Admiral George W. Calver, physician to Congress from 1928 to 1966, died February 27, 1972. He resided at 3835 Ellicott Street, N.W.

A native of Washington, Admiral Calver graduated from George Washington University medical school in 1912. Following his residency at Providence Hospital he entered the Naval Medical Reserve Corps in 1913 and in 1914 he was commissioned in the regular Naval Medical Corps. He served here and in the Pacific for 15 years before his assignment to Congress. Calver was a Lieutenant Commander assigned to the Washington Naval Dispensary in 1928 when he was appointed to be in attendance on the Sessions of Congress as the result of a resolution passed by the House of Representatives. In 1931 Congress passed legislation to prevent his transfer without congressional approval. He was promoted to Captain in 1936 and to Rear Admiral in 1945. He was placed on the retired list in 1947 after 33 years of service but continued on active duty status and was made a Vice Admiral in 1966.

A cardiologist, Admiral Calver worked unrelentingly to decrease the incidence of heart attacks and coronary deaths among congressmen. He introduced a "balanced metabolism routine" featuring a lean diet and daily exercise which he urged the legislators to follow.

Dr. Calver was an honorary lifetime member of the Medical Society of the District of Columbia. He was a fellow of the American College of Surgeons, the American College of Physicians and the American Geriatrics and Gerontological Societies. A Diplomate of the American Board of Internal Medicine, Admiral Calver was also a Fellow, Trustee and past President of the American College of Cardiology, past President of the Pan American Medical Society and a member of the American Medical Association.

He is survived by his wife, the former Jessie Willits; two daughters, Mrs. Paul F. Dickens, Jr. of Arlington and Mrs. Eldon C. Swanson of Florida; 10 grandchildren and two great-grandchildren.

Frank J. Eichenlaub, M.D.
1894–1972

Dr. Frank J. Eichenlaub, professor of dermatology at Georgetown University from 1928 to 1960, died February 13, 1972, after a heart attack at his home. He lived at 5217 Farrington Road, Westmoreland Hills, Maryland.

Dr. Eichenlaub was professor emeritus of dermatology at Georgetown and an emeritus consultant at Walter Reed General Hospital, and maintained a private practice in Washington, D. C. until his retirement in 1971. He was born in Erie, Pennsylvania in 1894. He received his B.S. degree in 1916 and his M.D. degree in 1918 from Georgetown University. He interned at St. Elizabeth's Hospital in Youngstown, Ohio, and took his dermatologic training in New York City at the Vanderbilt Clinic under the direction of the late Dr. George M. McKee. In 1922 he began the private practice of dermatology in Washington, D. C. with Dr. Henry Hazen. Dr. Eichenlaub, who helped establish the Outpatient Department at the old Georgetown Hospital, became a preceptor in dermatology and oversaw the postgraduate training of many resident physicians there and at Walter Reed.

He was the first to describe a rare tumor known as fibromyxoma, to which his name has been appended. He wrote many articles for medical journals.

He was a Diplomate of the American Board of Dermatology, a member of the American Academy of Dermatology; the American Dermatological Association, a life member of the Medical Society of the District of Columbia, the American Medical Association, of which he was past vice-president of the Section on Dermatology, the Osler Society, the Georgetown Clinical Society from which he received the "Man of the Year Award" in 1956, and the Southern Medical Association, of which he was a past president of the Section on Dermatology.

He leaves his wife, the former Dorothy Fisk; a daughter, Mrs. Jane E. Sunier of Rockville, two sons, John a physician who lives in Minneapolis, and Frank J., III, in Arizona, nine grandchildren and four great grandchildren.

By his colleagues in dermatology, Frank J. Eichenlaub will always be remembered for his unusual diagnostic acumen, which he was always willing to share with them. By his many graduate students, he will likewise be remembered as an excellent and most practical teacher who effectively combined the pathologic aspects of skin disease with the clinical side to present a truly complete knowledge of dermatology.

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> *"The history of science, and in particular the history of medicine . . . is . . . the history of man's reactions to the truth, the history of the gradual revelation of truth, the history of the gradual liberation of our minds from darkness and prejudice."*
>
> *—George Sarton, from "The History of Medicine Versus the History of Art"*

Should nongovernment scientists and physicians play a role in drug regulation?

Opinion

Results of a questionnaire to 7000 physicians:

82.8%
Physicians should play a role

78.3%
Independent scientists should play a role

69.8%
Medical academicians should play a role

Opinion &
Dialogue

Dialog

Should nongovernment scientists and physicians play a role in drug regulation?

Doctor of Medicine

Herbert L. Ley, Jr., M.D., M.P.H., Formerly Commissioner, F.D.A. (1968-1969) Currently Medical Consultant

In order for drug regulation to be effective, participation in the regulatory process from nongovernment physicians and scientists must be encouraged. Without such involvement, there will continue to be a high degree of controversy surrounding any regulations promulgated by the Food and Drug Administration.

There are two areas in which participation and communication by nongovernment physicians and scientists could significantly improve the process of regulation. First, scientists and physicians throughout the country could become involved in consulting relationships with the Food and Drug Administration in important scientific areas while regulatory policies are being evolved. If nongovernment professionals could bring their expertise and experience to bear early in the decision-making process, they would have less reason to criticize the final outcome.

Secondly, practicing physicians, academic physicians, and academic-based scientists could make it their business to comment on proposed regulations appearing in the Federal Register. Ideally, a system could be instituted whereby medical, scientific and technical people could see the Federal Register regularly, and provide the Food and Drug Administration with a body of opinion that has so far gone unheard. The FDA is caught among pressures from industry, Congress, the Presidential Administration and consumers. It should also feel pressures from practicing physicians and scientists.

In order to become more involved in these stages of the drug regulatory process, nongovernment physicians and scientists should begin to exercise their influence through their respective professional organizations, state and national medical societies, and specialty groups. Logically, a letter from these organizations representing a collective opinion has far greater weight in the regulatory process than individual letters. If the Food and Drug Administration receives opinions from these organizations early, before a regulation gets into the Federal Register, they are in a good position to respond with further study and review. Without such dissenting opinions, there is very little incentive to make changes in proposed regulations.

One instance in which practitioners did influence drug regulatory affairs in this way is the recent controversy that arose over the legitimacy of drug combinations. The strong opinion of practitioners on the value of such medication in clinical practice played a very prominent role in making the Food and Drug Administration modify its rather restrictive policy.

Another way in which practitioners can effectively influence drug regulations is by working with drug manufacturers conducting clinical trials of chemotherapeutic agents. When a drug is rated other than effective it may only mean that there is a lack of controlled clinical evidence as to efficacy. Thus, physicians might offer to conduct clinical studies that could help keep a truly effective drug in the marketplace. The treatment of diseases such as diabetes and angina are areas where the practitioner can aid in clinical studies because patients suffering from these diseases are rarely found in the conventional hospital setting.

By working with ethically and scientifically sound study designs in his everyday practice, the practitioner could begin to play an important part in determining official ratings on drug efficacy.

Nongovernment physicians and scientists and the FDA should also improve their lines of communication to the public. The medical community must develop a voice every bit as loud as that of the consumerists, the press, and others who sometimes criticize without complete information. If not, much of wha the medical communit *and* federal regulators d will often be represented i simplistic and somewha misleading terms.

One illustration of th misuse of the media in thi regard is the recall of anti coagulant drugs severa years ago. This FDA actio was given publicity by th press and television tha went far beyond its prob able importance. The resul was a very uncomfortabl situation for the practi tioner who had patient taking these medication Since the practitioner an pharmacist had not bee informed of the action b the time it was publicized in most states they wer deluged with calls fron worried patients.

The practitioner can at tempt to solve these prob lems of inadequate commu nication in several ways One would be the creatio of a communications lin in state pharmacy societies When drug regulation new is to be announced, the so ciety could immediatel distribute a message to ev ery pharmacist in the state The pharmacist, in turn could notify the physician in his local community s that he and the physicia could be prepared to an swer inquiries from pa tients. Another approach would be to use profes sional publications th practitioner receives.

All of this leads back t my opening contention: i drug regulation is to be ef fective, timely, and relate to the realities of clinica practice, a better method o communication and feedback must be developed between the nongovernmental medical and scientific communities and the regulatory agency.

One of a series

ue

Maker of Medicine

Henry W. Gadsden,
airman & Chief Executive
Officer, Merck & Co., Inc.

In my opinion, it is the sponsibility of all physicians and medical scientists t take whatever steps they tink are desirable in a law- d regulation-making ocess that can have far-aching impact on the actice of medicine. Yet any events in the recent st indicate that this is t happening. For example, it is apparent from ug efficacy studies that te NAS/NRC panels gave tle consideration to the e dence that could have ben provided by practicig physicians.

There are several current dvelopments that should icrease the concern of pacticing physicians about dug regulatory affairs. One is the proliferation of malpactice claims and litigatn. Another is the effort b government to establish te relative efficacy of dugs. This implies that if a physician prescribes a dug other than the "establihed" drug of choice, he my be accused of practicing something less than fst-class medicine. It wuld come perilously cse to federal direction of hw medicine should be pacticed.

In order to minimize this kd of arbitrary federal ation, a way must be fnd to give practitioners bth voice and representation in government affairs. Government must be caused to recognize the essentiality of seeking their views. One of the difficulties today, however, is that there is no way for concerned practitioners to participate in the early stages of decision-making processes. They usually don't hear about regulations until a proposal appears in the Federal Register, if then. By that time a lot of concrete has been poured, and a lot of boots are in the concrete.

Physicians in private practice, and particularly clinicians, should press for representation on the advisory committees of the Food and Drug Administration, joining with academic and teaching hospital physicians and scientists who are already serving. Though practitioners may not have access to all available information, the value of their clinical experience should be recognized. Clinicians, for example, rightly remind us that difficulty in *proving* precise effects does not necessarily mean a drug is ineffective.

Unless practitioners are more involved in drug regulations, it will be increasingly difficult for the pharmaceutical industry and scientists elsewhere to make optimal progress in drug development. The benefit/risk ratio must be re-emphasized, and as part of this it must be acknowledged that benefit can come from the judgments of medical science as a whole. Even this concept, unfortunately, is not always accepted in drug regulatory processes. For example, if current medical opinion holds that an excess of total lipids and cholesterol in the blood is probably predisposing to atherosclerosis, and if a drug is discovered which reduces total lipids and cholesterol, the drug ought to be accepted *prima facie* as a contribution to medical science . . . until someone disproves the theory. The sponsor should not have to prove the theory as well as to develop and test the drug.

I feel a major new effort must also be made to erase the feeling of mistrust of medicine and of medicines that seems to be growing in the public consciousness. Triggered primarily by strident announcements in Washington, people are reading and hearing confidence-shaking things almost continuously. Although challenge and awareness are essential to medical advancement, our long-term goal is constructively to build, not destroy. This means strengthening patient-physician relationships based on mutual confidence and trust. And in matters of health policy, it means working toward participatory rather than adversary proceedings—where everyone with an interest and a capacity to contribute has an opportunity to be heard . . . and, if that opportunity is not spontaneously afforded him, he may seek it.

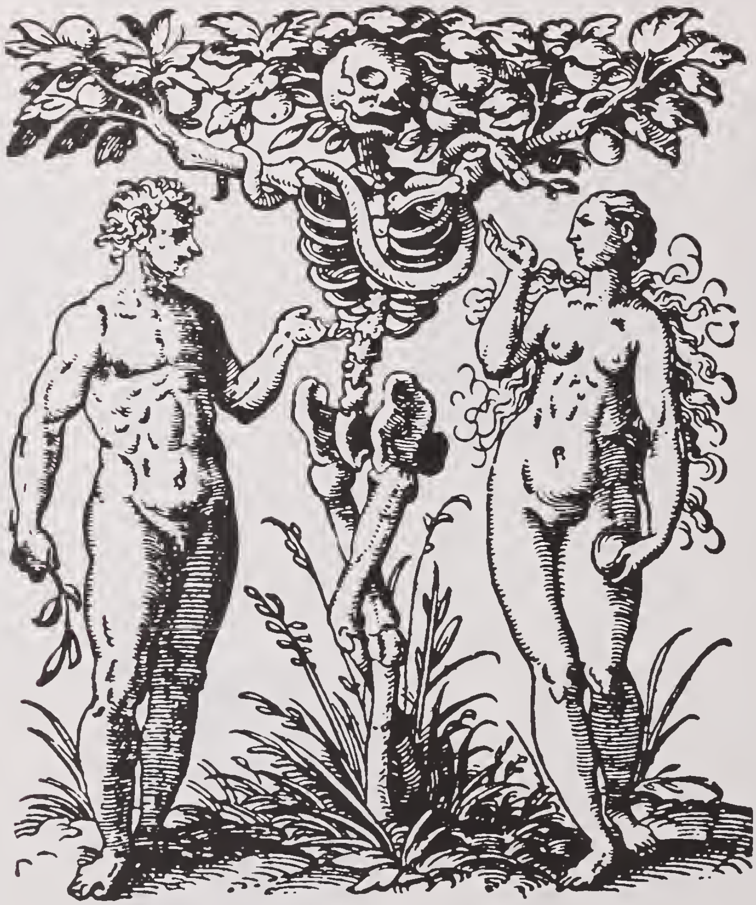
Opinion & Dialogue

What is your opinion, doctor?
We would welcome your comments.

P·M·A

The Pharmaceutical Manufacturers Association
1155 Fifteenth Street, N.W., Washington, D.C. 20005

In acute gonorrhea
(urethritis, cervicitis, proctitis when due to susceptible strains of N. gonorrhoeae)



Sterile Trobicin®
(spectinomycin dihydrochloride pentahydrate)—For Intramuscular injections, 2 gm vials containing 5 ml when reconstituted with diluent. 4 gm vials containing 10 ml when reconstituted with diluent.
An aminocyclitol antibiotic active *in vitro* against most strains of *Neisseria gonorrhoeae* (MIC 7.5 to 20 mcg/ml). Definitive *in vitro* studies have shown no cross resistance of *N. gonorrhoeae* between Trobicin and penicillin.

Indications: Acute gonorrheal urethritis and proctitis in the male and acute gonorrheal cervicitis and proctitis in the female when due to susceptible strains of *N. gonorrhoeae.*

Contraindications: Contraindicated in patients previously found hypersensitive to Trobicin. Not indicated for the treatment of syphilis.

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Warnings: Antibiotics used to treat gonorrhea may mask
delay the symptoms of incubating syphilis. Patients should
carefully examined and monthly serological follow-up fo
least 3 months should be instituted if the diagnosis of syphil
suspected.

Safety for use in infants, children and pregnant women has
been established.

Precautions: The usual precautions should be observed
atopic individuals. Clinical effectiveness should be monitore
detect evidence of development of resistance of *N. gonorrho*

Adverse reactions: The following reactions were obser
during the single-dose clinical trials: soreness at the injection
urticaria, dizziness, nausea, chills, fever and insomnia.
During multiple-dose subchronic tolerance studies in nor
human volunteers, the following were noted: a decrease in he

Trobicin®

sterile spectinomycin dihydrochloride pentahydrate, Upjohn

single-dose intramuscular treatment

High cure rate:* 96% of 571 males, 95% of 294 females
Dosages, sites of infection, and criteria for diagnosis and cure are defined below.)**

Assurance of a single-dose, physician-controlled treatment schedule

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No allergic reactions occurred in patients with an alleged history of penicillin sensitivity when treated with Trobicin, although penicillin antibody studies were not performed

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Active against most strains of Neisseria gonorrhoeae in vitro (M.I.C. 7.5-20 mcg/ml)

A single two-gram injection produces peak serum concentrations averaging about 100 mcg/ml in one hour (average serum concentrations of 15 mcg/ml present 8 hours after dosing)

Note: Antibiotics used in high doses for short periods of time to treat gonorrhea may mask or delay the symptoms of incubating syphilis. Since the treatment of syphilis demands prolonged therapy with any effective antibiotic, and since Trobicin is not indicated in the treatment of syphilis, patients being treated for gonorrhea should be closely observed clinically. Monthly serological follow-up for at least 3 months should be instituted if the diagnosis of syphilis is suspected. Trobicin is contraindicated in patients previously found hypersensitive to it.

Data compiled from reports of 14 investigators. **Diagnosis was confirmed by cultural identitication of *N. gonorrhoeae* on Thayer-Martin media in all patients. Criteria for cure: negative culture after at least 2 days post-treatment in males and at least 7 days post-treatment in females. Any positive culture obtained post-treatment was considered evidence of treatment failure even though the follow-up period might have been less than the periods cited above under "criteria for cure" except when the investigator determined that reinfection through additional sexual contacts was likely. Such cases were judged to be reinfections rather than relapses or failures. These cases were regarded as non-evaluatable and were not included. JA72 1848-6

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lobin, hematocrit and creatinine clearance; elevation of alkane phosphatase, BUN and SGPT. In single and multiple-dose tudies in normal volunteers, a reduction in urine output was oted. Extensive renal function studies demonstrated no conistent changes indicative of renal toxicity.

Dosage and administration: Keep at 25°C and use within 4 hours after reconstitution with diluent.
Male—single 2 gram dose (5 ml) intramuscularly. Patients with onorrheal proctitis and patients being re-treated after failure f previous antibiotic therapy should receive 4 grams (10 ml). In eographic areas where antibiotic resistance is known to be prealent, initiol treatment with 4 grams (10 ml) intramuscularly is referred.
emale—single 4 gram dose (10 ml) intramuscularly.

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Susceptibility Powder—for testing *in vitro* susceptibility of *N. gonorrhoeae*.

Human pharmacology: Rapidly absorbed after intramuscular injection. A two-gram injection produces peak serum concentrations averaging about 100 mcg/ml at one hour with 15 mcg/ml at 8 hours. A four-gram injection produces peak serum concentrations averaging 160 mcg/ml at two hours with 31 mcg/ml at 8 hours.
For additional product information, see your Upjohn representative or consult the package insert. MED-B I S (LWB)

Upjohn The Upjohn Company, Kalamazoo, Michigan 49001

Pre-Sate® (chlorphentermine hydrochloride)

Caution: Federal law prohibits dispensing without prescription.

Indications

Pre-Sate (chlorphentermine hydrochloride) is indicated in exogenous obesity, as a short term (i.e. several weeks) adjunct in a regimen of weight reduction based upon caloric restriction.

Contraindications

Glaucoma, hyperthyroidism, pheochromocytoma, hypersensitivity to sympathomimetic amines, and agitated states. Pre-Sate (chlorphentermine hydrochloride) is also contraindicated in patients with a history of drug abuse or symptomatic cardiovascular disease of the following types: advanced arteriosclerosis, severe coronary artery disease, moderate to severe hypertension, or cardiac conduction abnormalities with danger of arrhythmias. The drug is also contraindicated during or within 14 days following administration of monamine oxidase inhibitors, since hypertensive crises may result.

Warnings

When weight loss is unsatisfactory the recommended dosage should not be increased in an attempt to obtain increased anorexigenic effect; discontinue the drug. Tolerance to the anorectic effect may develop. Drowsiness or stimulation may occur and may impair ability to engage in potentially hazardous activities such as operating machinery, driving a motor vehicle, or performing tasks requiring precision work or critical judgment. Therefore, such patients should be cautioned accordingly. Caution must be exercised if Pre-Sate (chlorphentermine hydrochloride) is used concomitantly with other central nervous system stimulants. There have been reports of pulmonary hypertension in patients who received related drugs.

Drug Dependence Drugs of this type have a potential for abuse. Patients have been known to increase the intake of drugs of this type to many times the dosages recommended. In long-term controlled studies with the high dosages of Pre-Sate, abrupt cessation did not result in symptoms of withdrawal.

Usage In Pregnancy The safety of Pre-Sate (chlorphentermine hydrochloride) in human pregnancy has not yet been clearly established. The use of anorectic agents by women who are or who may become pregnant, and especially those in the first trimester of pregnancy, requires that the potential benefit be weighed against the possible hazard to mother and child. Use of the drug during lactation is not recommended. Mammalian reproductive and teratogenic studies with high multiples of the human dose have been negative.

Usage In Children Not recommended for use in children under 12 years of age.

Precautions

In patients with diabetes mellitus there may be alteration of insulin requirements due to dietary restrictions and weight loss. Pre-Sate (chlorphentermine hydrochloride) should be used with caution when obesity complicates the management of patients with mild to moderate cardiovascular disease or diabetes mellitus, and only when dietary restriction alone has been unsuccessful in achieving desired weight reduction. In prescribing this drug for obese patients in whom it is undesirable to introduce CNS stimulation or pressor effect, the physician should be alert to the individual who may be overly sensitive to this drug. Psychologic disturbances have been reported in patients who concomitantly receive an anorectic agent and a restrictive dietary regimen.

Adverse Reactions

Central Nervous System: When CNS side effects occur, they are most often manifested as drowsiness or sedation or overstimulation and restlessness. Insomnia, dizziness, headache, euphoria, dysphoria, and tremor may also occur. Psychotic episodes, although rare, have been noted even at recommended doses. **Cardiovascular:** tachycardia, palpitation, elevation of blood pressure. **Gastrointestinal:** nausea and vomiting, diarrhea, unpleasant taste, constipation. **Endocrine:** changes in libido, impotence. **Autonomic:** dryness of mouth, sweating, mydriasis. **Allergic:** urticaria. **Genitourinary:** diuresis and, rarely, difficulty in initiating micturition. **Others:** Paresthesias, sural spasms.

Dosage and Administration

The recommended adult daily dose of Pre-Sate (chlorphentermine hydrochloride) is one tablet (equivalent to 65 mg chlorphentermine base) taken after the first meal of the day. Use in children under 12 not recommended.

Overdosage

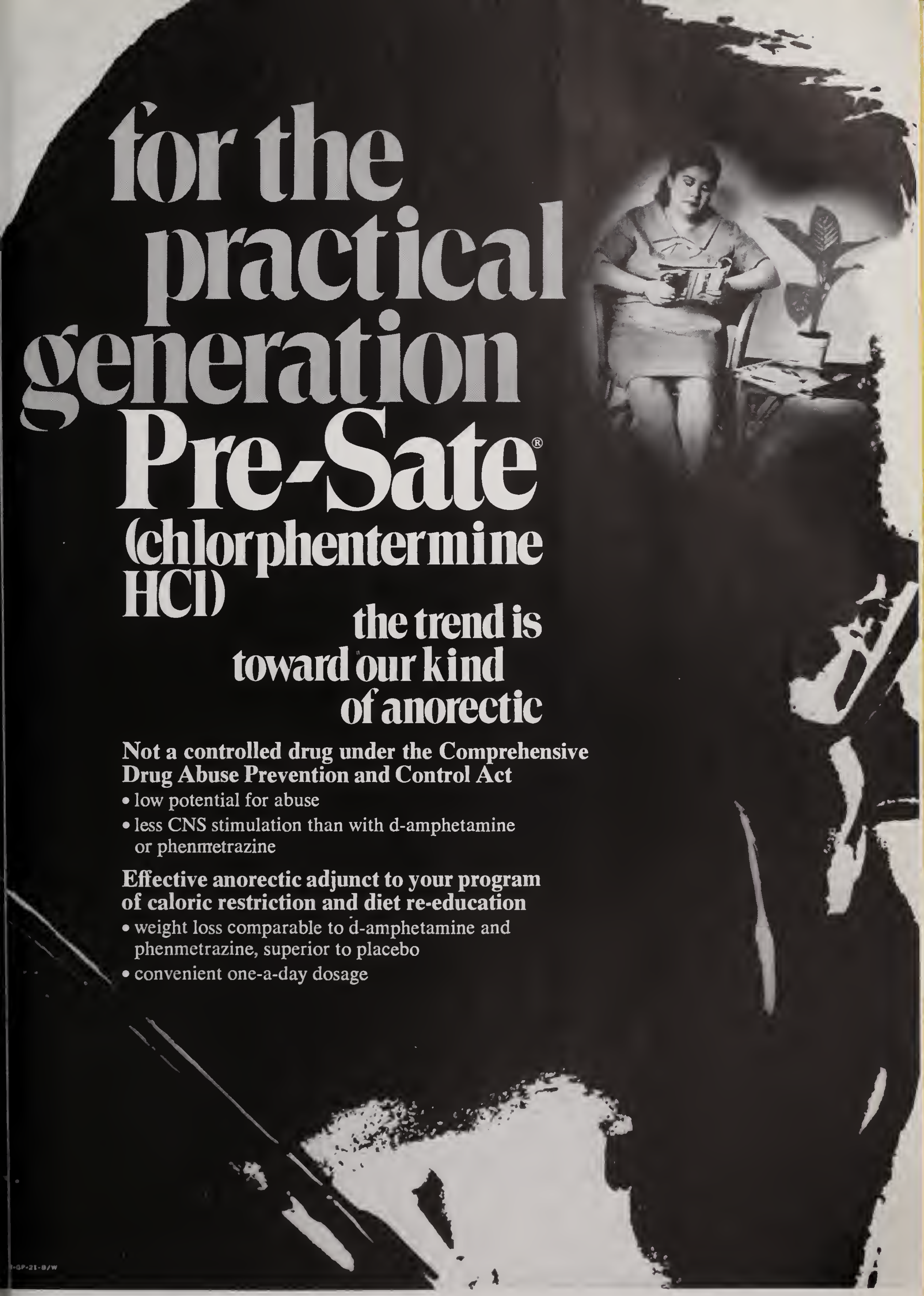
Manifestations: Restlessness, confusion, assaultiveness, hallucinations, panic states, and hyperpyrexia may be manifestations of acute intoxication with anorectic agents. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension, or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Fatal poisoning usually terminates in convulsions and coma.

Management: Management of acute intoxication with sympathomimetic amines is largely symptomatic and supportive and often includes sedation with a barbiturate. If hypertension is marked, the use of a nitrate or rapidly acting alpha-receptor blocking agent should be considered. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendations in this regard.

How Supplied

Each Pre-Sate (chlorphentermine hydrochloride) tablet contains the equivalent of 65 mg chlorphentermine base; bottles of 100 and 1000 tablets.

Full information is available on request.



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(chlorphentermine HCl)

the trend is toward our kind of anorectic

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Effective anorectic adjunct to your program of caloric restriction and diet re-education

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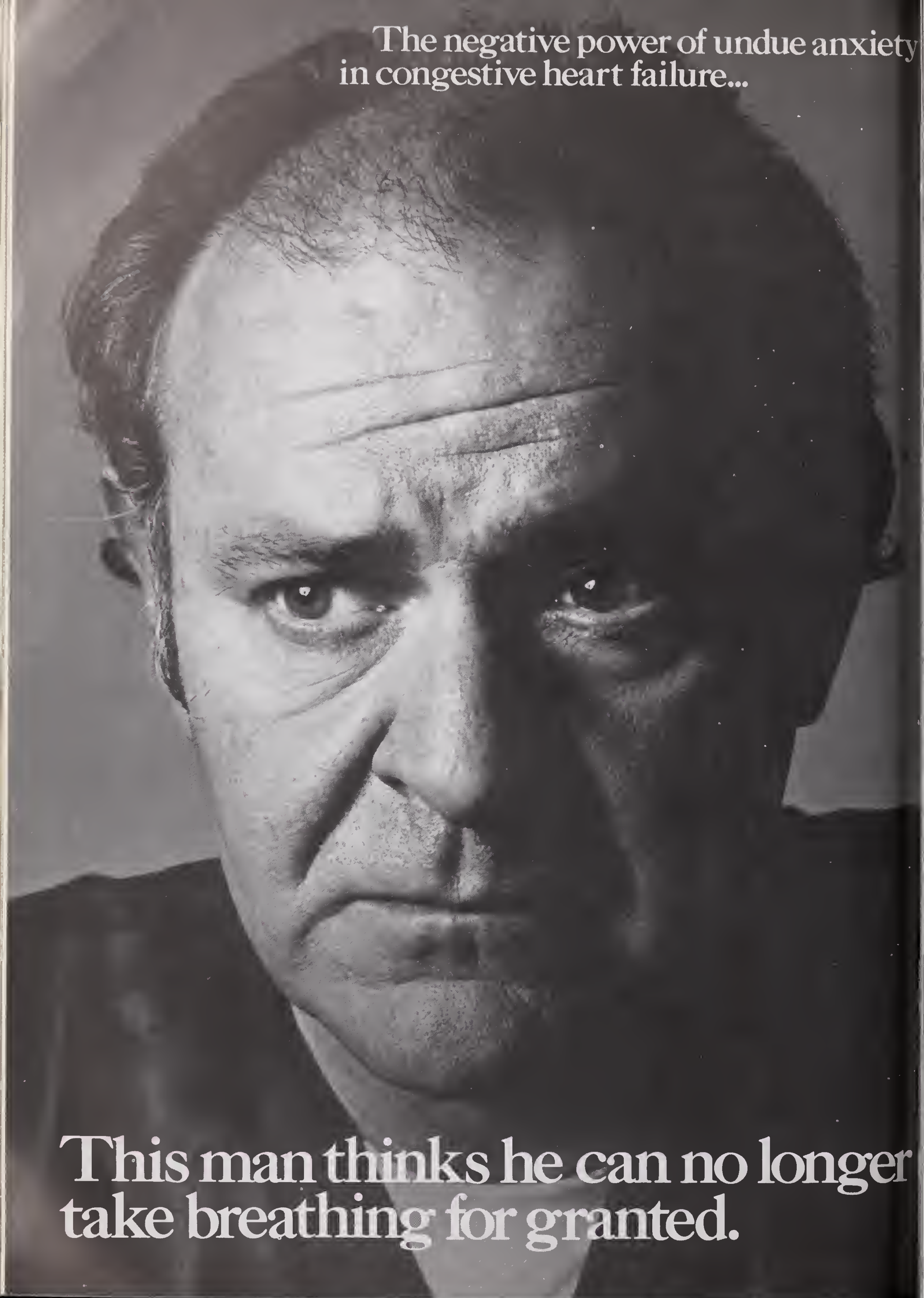
- convenient one-a-day dosage

GP-21-B/W

Pre-Sate® (chlorphentermine HCl)...the increasingly practical appetite suppressant


The negative power of undue anxiety in congestive heart failure...
This man thinks he can no longer take breathing for granted.

Typical of many patients with congestive heart failure, he also suffers from severe anxiety, a psychic factor that may influence the character and degree of his symptoms, such as dyspnea. His apprehension may also deprive him of the emotional calm so important in maintenance therapy.

Aid in rehabilitation
Specific medical and environmental measures are often enhanced by the antianxiety action of adjunctive Libritabs (chlordiazepoxide). Libritabs can also facilitate treatment of the tense convalescent patient until antianxiety therapy is no longer required. Whereas in geriatrics the *usual daily dosage* is 5 mg two to four times daily, the *initial dosage* in elderly and debilitated patients should be limited to 10 mg or less per day, adjusting as needed and tolerated.

Concomitant use with primary agents
Libritabs is used concomitantly with certain specific medications of other classes of drugs, such as cardiac glycosides, diuretics, antihypertensives, vasodilators and oral anticoagulants, whenever excessive anxiety or emotional tension adversely affects the clinical condition or response to therapy. Although clinical studies have not established a cause and effect relationship, physicians should be aware that variable effects on blood coagulation have been reported very rarely in patients receiving oral anticoagulants and chlordiazepoxide HCl.

The positive power of

Libritabs®

(chlordiazepoxide)

5-mg, 10-mg, 25-mg tablets

t.i.d./q.i.d.

up to 100 mg daily

for severe anxiety accompanying congestive heart failure

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Indicated when anxiety, tension and apprehension are significant components of the clinical profile.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported. Use of any drug in pregnancy, lactation, or in women of childbearing age requires that its potential benefits be weighed against its possible hazards.

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Supplied: Tablets containing 5 mg, 10 mg or 25 mg chlordiazepoxide.

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FOR SALE: An Ophthalmologist and Otolaryngologist is retiring and will sell contents of his office and records with option to lease office. Good location, upper Connecticut, plenty of parking, especially suitable for psychiatrist, eye or nose and throat specialist. Telephone WO-6-1323.

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The Clinical Utility of In-Vitro Tests of Thyroid Function —
Jay R. Shapiro, M.D. and
Richard C. Reba, M.D.

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PRESIDENT'S MESSAGE

GO GREENBRIER

If you have not reviewed the program of the Society's Annual Scientific Assembly recently mailed to all members, take five minutes to look at it right now. Note particularly:

1. Six in-depth "seminar work shops" will replace the former series of unrelated lectures. Three of these will explore "contemporary" topics; three, scientific ones. It is contemplated that members will sign up to attend a single workshop all three days, but "cross over" will be permitted.
2. Every seminar presents persons of national reputation, knowledgeable in the particular field, and skillful in the art of teaching.
3. Wives are invited to participate in the contemporary seminars.
4. Paramedical personnel are invited to participate also.
5. A single one-hour plenary session will begin each day's program.
6. A prayer breakfast will be held Sunday morning.
7. The scientific program ends at 12 noon each day.
8. The afternoon sports program includes:
    - a. A men's golf tournament
    - b. A women's golf tournament
    - c. A tennis tournament
    - d. A trap shooting contest

But all the attractions could not be crowded into the program, and many deserve mention.

rrell C. Crain, M.D.

1. The Greenbrier is one of the outstanding resort hotels in this country, its decor by Dorothy Draper presenting a sophistication of color combinations unmatched anywhere in the world, and its cuisine an unusual presentation of the finest foods available.
2. The Arts and Crafts Colony on the Greenbrier campus is an intriguing group of shops and demonstrations particularly attractive to the ladies.

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ABDICATE NOT FROM THIS RESPONSIBILITY!

Costs of health care delivery continue to soar. One of the expected results of this fact is the increased payments demanded of the health insurance carriers. The carrier can only respond in one of two ways - increase premiums or reduce coverage. In this regard most of the commercial carriers have elected to follow the lead taken by the Social Security Administration in its management of the Medicare program. There has been an increase in the Medicare "premium," but a far more politically acceptable step has been the reduction of coverage. As all physicians involved in primary medical care of patients know, there has been a severe cutback in extended care post-hospitalization coverage. Indeed, in a number of good quality extended care nursing facilities in the metropolitan area, where a sizeable portion of the patient population formerly was under Medicare coverage, a patient so covered presently in these institutions is a rarity.

Certainly this has reduced the costs of the Medicare program but unfortunately it has undoubtedly reduced the quality of total patient care and further has certainly tended to elongate patient stays in acute care institutions where the day-to-day costs continue at a much higher level. Aware of this latter fact, the insurance companies sub-contracted to administer the Medicare program have begun increasingly intense surveillance of acute care hospital charts. This is generally being accomplished by physicians in the employ of the carrier, and at least to date this review task has not been assigned to clerical personnel with computer assistance. However, this is obviously a form of unilateral, external bed utilization review. A popular topic of conversation these days in certain quarters is the court case recently decided in Philadelphia where a judgement was rendered against a physician requiring him to pay a portion of the patient's hospital bill. Astonishing, you say, but a fact and an indication most likely of more to come.

Bed utilization review has been an established mechanism in almost all hospitals for several years now. Much paper work and verbiage has resulted, but little effective pressure has been generated to improve bed utilization and reduce the length of hospital stays. The latter has been reduced in most local institutions, but I submit that this has happened because of increased demands for beds and longer waiting lists for admission largely because of health care insurance coverage (Medicaid) for those lower income groups previously not covered.

What is wrong with the Bed Utilization Review Committees in our hospitals? Why can't these committees be more effective, more forceful? How can the initiative be regained by the medical profession and the hospitals? Unfortunately, these committees were originally established because it was a requirement of the health care legislation of the 1960's. But these committees were established with emphasis on the word review; no powers were vested in the charges of these committees, and thus today the chairmen and members of these Utilization Review Committees often flounder, relegated to recommendations or pleadings, deprived from making firm judgements. Much of this, of course, is in the tradition of American medicine. We have a very poor record of self-policing and self-discipline. And therefore, here is another example of where the social and financial pressures of today are causing controls to emanate from outside the system, that is from the insurance companies, with the federal government calling the shots.

Why can't the medical staffs and administrative staffs of hospitals, working in concert, put teeth into utilization review? If done, costs could be reduced, and extended care coverage again might become available to patients without excessive and unrealistic financial drain. The hour grows late in many areas of the health care system. Utilization review is but one example of where physicians must become agressive and effective in self-policing. Because, after all, who can do the job better than physicians. Abdicate not from this responsibility!

Louis Gillespie, Jr., M.D.

"Everybody talkin' 'bout Jesus and Mary
Nobody talkin' 'bout Joe, poor Joe."(1)

"Everybody talkin' 'bout heroin and hash
Nobody talkin' 'bout booze."(2)

With close to 10 million alcoholics in the United States, alcoholism in sheer numbers, dollar loss, crime production, and psycho-social devastation is by far this country's number one drug problem. Yet, in spite of the enormity, little attention is being paid this problem, not only by the public, but by organized medicine as well. Ninety-five per cent of this country's alcoholic individuals appear as "ordinary citizens"; only five per cent are seen in our offices for complications of their addiction. Because of our attention to the medical and surgical complications, rather than to the disease of alcoholism itself, we have not been able to apply the principles of early diagnosis, treatment, and preventive medicine measures to the latter. All practicing physicians, regardless of specialty, see patients who are alcoholics; these physicians are capable of, and should be, treating the alcoholic. Although there are many medical specialists who deal exclusively in the treatment of alcoholism, the first line of defense is recognition and treatment of the disease process by the first physician to see the patient.

It is the very strong opinion of the Committee on Alcoholism of the Medical Society of D.C., as well as that of numerous recovered alcoholics (yes, it is a treatable, reversible and controllable disease) that the number one duty facing the physician with respect to alcohololgy is to learn more about the disease itself rather than just its complications.

This can be accomplished by many means, one of which is post-graduate education. In a forthcoming article, an in-depth view of the early recognition, diagnosis, approach, and treatment of the alcoholic prior to the development of medical complications will be undertaken. By so arming the medical community with information which has been so neglected by many of our medical school curricula, it is hoped that more people will have their disease recognized earlier and obtain medical treatment sooner than in the past.

Howard M. Silby, M.D.

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SHORTAGE OF PARAMEDICAL PERSONNEL

Improvement of health care delivery systems is dependent on a number of important factors. One is available paramedical manpower. Practically every physician is well aware that there are not sufficient numbers of allied health personnel. Not only does this exist today, it is apt to become even more critical in the future. This situation has prompted a number of study programs sponsored by government agencies and non-governmental organizations. One such program was made possible by the Manpower Administration of the U.S. Department of Labor in 1970. Another was supported by the NIH Division of Allied Health Manpower. Further, the Office of Education of HEW attempted to stabilize the various education programs for new types of health occupations, by declaring a moratorium on accreditation of new school programs pending a national study sponsored by the American Medical Association's Council on Medical Education, the National Commission of Accreditation and the Association of Schools of Allied Health Professions. Their report was submitted to Congress by the Secretary of HEW. Entitled, "A Report on Licensure and Related Health Personnel Credentialing," it identified major problems associated with licensure, certification and other qualifications for practice or employment of health personnel, together with the summaries of the activities of federal agencies, professional organizations and other instrumentalities directed toward maximizing the proper and efficient utilization of health personnel in meeting the health needs of the nation.

A similar report, dealing with educational activities in areas allied with medicine, was published in JAMA in the "Education Number," Volume 28, No. 8, November 22, 1971.

Despite all of the attention, interest, efforts, funding and meetings the problem still exists. There is a shortage of allied medical personnel and it is getting worse, not better. Evaluation of data released by the Bureau of Labor Statistics shows that there are roughly 86,284,000 people in the civilian labor force and that the number of unemployed persons is somewhere around 5,000,000. The change in the unemployment rate through the first months of 1972 has been very significant to the statistician and the politician, but not to the public. Furthermore, in June 1972 approximately

1,000,000 young people will be graduated from educational institutions and will enter the labor force in some capacity. These people will not necessarily get integrated into health science fields where they are needed. With the reduction of military manpower, the available work force will tend to increase. Since there is a need for people in the allied health field and there is an increase in the available manpower pool, it seems reasonable that we should be able to overcome the shortage of paramedical personnel as the consumer demands for medical service increase.

The federal government works in strange ways and these do not necessarily lead to the solution of these problems today and maybe not even in the future. Meanwhile, the shortage of paramedical personnel marches on. An example of how the "left hand" of the federal government does not know what the "right hand" of the federal government is doing in solving the problem of allied health manpower, the U.S. Department of Labor is working hard with some branches of HEW to recruit, motivate and train individuals to enter the paramedical health field. These programs do not necessarily rely on the formal education programs conducted by universities, colleges or medical institutions. The objective is to produce highly skilled, skilled, and semiskilled paramedical personnel. They are attempting to develop alternate ways to utilize corpsmen and paramedical technicians from the military services as well as individuals who have attended schools not professionally accredited or who can respond to on-the-job training. Furthermore, they are considering ways to improve opportunities for these people to progress up the career ladder without formal education or with limited formal education. They are also considering retraining people who have retired from the field and now wish to re-enter.

The Manpower Administration has gone so far as to back the development of proficiency examinations to assess the knowledge and skills of aspiring clinical laboratory personnel who do not have formal education or certification under present existing jurisdiction. This all looks very encouraging until one sees other branches of HEW which stand in opposition, perhaps unintentionally, to the entrance and employment of individuals with certain levels of training, informal or formal education, into paramedical health sciences - notably, the Social Security Administration through the rules and regulations of the Medicare law and the

National Center for Disease Control under the rules and regulations of the Clinical Laboratories Improvement Act. Both justify their position in that they must control the quality performance of medical and paramedical personnel. The net effect of this is to preclude the admission of skilled and semi-skilled paramedical personnel into the manpower pool of highly skilled personnel. A puzzling situation.

William J. Jaffurs, M.D.

DOCPAC

The two questions I encounter most frequently about DOCPAC are: 1) What is it? and 2) What does it do?

ANSWER 1: DOCPAC is the Political Action Committee of the District of Columbia. It is a component part of AMPAC, the American Medical Political Action Committee. Anyone joining DOCPAC automatically becomes a member of AMPAC. Single membership is $20.00 per year, family membership is $30.00 per year while sustaining membership is $100.00 per year.

ANSWER 2: AMPAC, through DOCPAC and 49 other component organizations is concerned with the selection and election of public officials whose political attitudes on medical matters are in reasonable harmony with the philosophy of organized medicine. This involves an appreciation of our system of private practice and free enterprise, coupled with a desire to improve the system without destroying it.

Obviously, the election of sympathetic congressmen and senators constitutes the most effective form of lobbying; and in this effort, AMPAC has achieved recognition as one of the most effective action groups in the country. In the election of 1970, over 75 per cent of the AMPAC-backed candidates were elected.

In the District, DOCPAC, through a Candidate Evaluation Committee last year established a close relationship with both major candidates, Reverend Walter Fauntroy, who was elected District Delegate to Congress, and John Nevius, who is now Chairman of the City Council. In this way, the interest of physicians in such important legislation as the Professional Incorporation Act (which was enacted by Congress in 1971), the Healing Arts Practice Act, the Medical Records Bill and Blood Bank legislation can be brought to the attention of Congress effectively.

The year 1972 and each succeeding year will be vital to the cause of American medicine as Congress moves closer to the enactment of some form of sweeping health legislation. The shape of that legislation will depend on the makeup of Congress which in turn depends on the effective support of the right candidates.

Will you join DOCPAC and AMPAC today so that your voice can be heard where it counts? Send your check to DOCPAC, in care of the Medical Society of D.C., 2007 Eye Street, N.W., Washington, D.C. 20006.

Can you do less?

Karl C. Jonas, M.D.
Chairman, DOCPAC

THE 7-DAY WEEK

It has occurred to many of us on occasion how much time is wasted in hospitals by the traditional weekend lack of service.

Diagnostic roentgenograms, especially involving contrast studies and fluoroscopy, are unavailable after noon on Saturdays, except in an emergency. Certain laboratory studies also fall into this category, with some variation from hospital to hospital. Perhaps the greatest waste is in the use of the operating room, which is usually restricted to emergency operations on Saturdays and Sundays. It is not infrequent for a patient to wait in the hospital over a weekend in order to undergo an elective operation on Monday.

Where lies the fault? It seems unlikely that our local hospital administrators have not considered this idea in an effort to reduce the length of a patient's hospital stay. It has been tried successfully in other parts of the country.

It is up to us as practicing physicians to promote this concept, if we agree it has virtue. Implementation should not be insurmountable. It only requires a change in attitude of many physicians and administrators. After all, the week does have seven days.

Jerome Canter, M.D.

140/90 is normal blood pressure…or is it?

An extensive study based on nearly 4 million life insurance policies suggests that a blood pressure reading of 140/90 requires close medical supervision.

The Build
and
Blood Pressure
Study

Study Findings. Twelve years ago he Society of Actuaries reported on an extensive study based on the lives and deaths represented by almost 4 million life insurance policies. From this vast survey—"The Build and Blood Pressure Study"[1]—insurance experts concluded that:

- Blood pressure above 140/90 is accompanied by increased morbidty and requires close medical attention.
- Even small increments in either systolic or diastolic blood pressure progressively and steeply shorten ife expectancy.

Other Studies. Studies conducted with large numbers of patients since that time have echoed the above findings. Two studies published in 1970—the VA Cooperative Study Group on "Effects of Treatment on Morbidity in Hypertension"[2] and the "Framingham Study"[3]—suggest that treatment of even mild hypertension may, over time, offer significant benefits to the patient.

Another Point of View. Although a growing body of studies suggests that treatment of mild hypertension is warranted, medical opinion is not unanimous. Some clinicians recommend that drug treatment for mild hypertension be reserved for patients with additional risk factors such as smoking, high cholesterol levels, heart or kidney involvement, or a family history of vascular disease. Dr. Walter M. Kirkendall stated this position in his recent paper "What's With Hypertension These Days?"[4] Discussing the management of hypertension in patients with a sustained diastolic pressure up to 100 mm Hg, he said: "Generally, I do not recommend antihypertensive therapy unless patient's blood pressure approaches the upper limit for the group and a number of adverse factors exist, such as male sex, family history of vascular disease, youth, evidence of heart or kidney involvement."

Drug Therapy for Hypertension. Although opinion varies on when to start drug therapy for mild hypertension, many physicians agree that treatment should start with a thiazide diuretic such as HydroDIURIL. For the adult patient, the usual starting dosage is 50 mg b.i.d. Dosage adjustments are recommended as the patient responds to treatment. The patient whose therapy begins with HydroDIURIL frequently can continue to benefit from it, because HydroDIURIL usually maintains its antihypertensive effect even when therapy is prolonged.

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Therapy to Start With

For a brief summary of prescribing information, please see next page.

[1] Society of Actuaries, *The Build and Blood Pressure Study*, 1959.

[2] Veterans Administration Cooperative Study Group on Antihypertensive Agents, "Effects of Treatment on Morbidity in Hypertension," JAMA *213*:1143-1152, Aug. 17, 1970.

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[4] Kirkendall, Walter M.: "What's With Hypertension These Days?" Consultant, Jan. 1971.

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CONTRAINDICATIONS: Anuria; increasing azotemia and oliguria during treatment of severe progressive renal disease. Known sensitivity to this compound. Nursing mothers; if use of drug is deemed essential, patient should stop nursing.

WARNINGS: May precipitate or increase azotemia. Use special caution in impaired renal function to avoid cumulative or toxic effects. Minor alterations of fluid and electrolyte balance may precipitate coma in hepatic cirrhosis.

When used with other antihypertensive drugs, careful observation for changes in blood pressure must be made, especially during initial therapy. Dosage of other antihypertensive agents, especially ganglion blockers, must be reduced by at least 50% because HydroDIURIL potentiates their action.

Stenosis and ulceration of the small bowel causing obstruction, hemorrhage, and perforation have been reported with the use of enteric-coated potassium tablets, either alone or with nonenteric-coated thiazides. Surgery was frequently required, and deaths have occurred. Such formulations should be used only when indicated and when dietary supplementation is impractical. Discontinue immediately if abdominal pain, distention, nausea, vomiting, or gastrointestinal bleeding occurs.

Thiazides cross placenta and appear in cord blood. In women of childbearing age, potential benefits must be weighed against possible hazards to fetus, such as fetal or neonatal jaundice, thrombocytopenia, and possibly other adverse reactions which have occurred in the adult.

The possibility of sensitivity reactions should be considered in patients with a history of allergy or bronchial asthma. The possibility of exacerbation or activation of systemic lupus erythematosus has been reported for sulfonamide derivatives, including thiazides.

PRECAUTIONS: Check for signs of fluid and electrolyte imbalance, particularly if vomiting is excessive or patient is receiving parenteral fluids. Warning signs, irrespective of cause, are dryness of mouth, thirst, weakness, lethargy, drowsiness, restlessness, muscle pains or cramps, muscular fatigue, hypotension, oliguria, tachycardia, and gastrointestinal disturbances. Hypokalemia may develop (especially with brisk diuresis) in severe cirrhosis; with concomitant steroid or ACTH therapy; or with inadequate electrolyte intake. Digitalis therapy may exaggerate metabolic effects of hypokalemia, especially with reference to myocardial activity. Hypokalemia may be avoided treated by use of potassium chloride or giving foo with a high potassium content. Similarly, any chlori deficit may be corrected by use of ammonium chlori (except in patients with hepatic disease) and large prevented by a near normal salt intake. Hypochlorem alkalosis occurs infrequently and is rarely severe. severely edematous patients with congestive failure renal disease, a low salt syndrome may occur if dieta salt is unduly restricted, especially during hot weathe

Thiazides may increase responsiveness to tuboc rarine. The antihypertensive effect of the drug may enhanced in the postsympathectomy patient. Arteri responsiveness to norepinephrine is decreased, nece sitating care in surgical patients. Discontinue drug hours before elective surgery. Orthostatic hypotensic may occur and may be potentiated by alcohol, barbi urates, or narcotics.

Pathological changes in the parathyroid glands wi hypercalcemia and hypophosphatemia have been see in a few patients on prolonged thiazide therapy. Th effect of discontinuing thiazide therapy on serum ca cium and phosphorus levels may be helpful in asses ing the need for parathyroid surgery in such patient Parathyroidectomy has elicited subjective clinical in provement in most patients, but has no effect o hypertension. Thiazide therapy may be resumed aft surgery.

Use cautiously in hyperuricemic or gouty patient gout may be precipitated. May affect insulin requir ments in diabetics; may induce hyperglycemia ar glycosuria in latent diabetics.

ADVERSE REACTIONS: Rare reactions inclu thrombocytopenia, leukopenia, agranulocytosis, apla tic anemia, cholestasis, and pericholangiolitic hepatiti Nausea, vomiting, diarrhea, dizziness, vertigo, pare thesias, transient blurred vision, sialadenitis, purpur rash, urticaria, photosensitivity, or other hypersens tivity reactions may occur. Cutaneous vasculitis pr cipitated by thiazide diuretics has been reported elderly patients on repeated and continuing exposu to several drugs. Scattered reports have linke thiazides to pancreatitis, xanthopsia, neonatal thron bocytopenia, and neonatal jaundice. When adver reactions are moderate or severe, the dosage thiazides should be reduced or therapy withdraw

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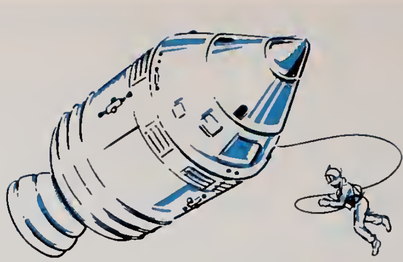

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>
> *—George Sarton, from "The History of Medicine Versus the History of Art"*

Would it be useful in clinical practice to have government predetermine drugs of choice?

Opinion

Results of a survey of physicians:

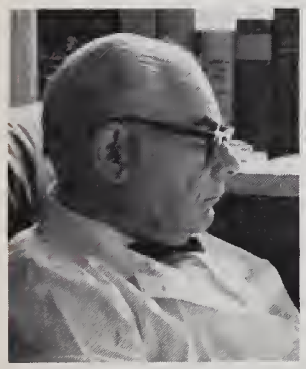
13.3%
Yes, it would be useful.

86.7%
No, it would not be useful.

Would it be useful in clinical practic to have government predetermine drugs of choice?

Doctor of Medicine

Walter Modell, M.D.,
Professor of Pharmacology,
Cornell University
Medical College,
Editor,
Clinical Pharmacology
& Therapeutics,
Drugs of Choice,
Rational Drug Therapy



The proposition that government should determine one or two "drugs of choice" within a given therapeutic class reflects the belief that a similarity in molecular structure insures a close similarity in pharmacologic effect. But this is by no means the rule. An obvious example would be in the field of diuretics, where a small change in chemical structure accounts for substantial differences in concomitant effects such as potassium excretion.

Any attempt to dictate the "drug of choice" would be complicated by the fact that some populations demonstrate a bimodal distribution in their reaction to drugs. If the data on drug response are mixed for the total population, one drug will appear to be as useful as the other. But if drug response is reported separately for different segments of the population, drug A will be found to be better for one group and drug B for the other.

It may, of course, be possible to determine drugs of choice in particular categories on a broad statistical basis. But there are always certain patients in whom a drug produces odd, unpredictable or idiosyncratic reactions. So, though a drug might statistically be the most useful one in a given situation, individual variations in response might make it the *incorrect* one.

The point I wish to make is that if two, three, four or more drugs in one class are of approximately equal merit, that in itself is justification for their availability. Exceptional cases do arise in which one drug would be useful to a certain segment of the population and another drug would be of no use at all. In the practice of medicine, the physician must be prepared to treat the routine as well as the unusual case.

Another objection to the determination of a drug of choice is that precise statements of *relative* efficacy are very difficult to make—much more difficult than statements of efficacy. For example, in testing drug efficacy, it is easy to determine the difference between a drug that is effective in treating a condition and one that is not at all effective. Thus, it is fairly easy to determine whether a drug is more effective than a placebo. But if you compare one drug that is effective with another drug that is also effective, and the relative differences between them are very slight, statements of relative efficacy may be very difficult to make with assurance.

I do not mean to imply that relative efficacy statements are not useful or can never be made. With some groups of drugs (e.g., analgesics), extensive study and precise methodology have yielded useful information on relative efficacy. But in most situations, such information can be acquired only through studies encompassing three to five years of use in many more patients than are used to compare drugs with a placebo for the introduction of a drug into commerce. It is really only after practitioners use a drug extensively that relative safety and efficacy in practice can really determined.

The Bureau of Drugs suggested the package sert as a possible mean communicating informa on relative efficacy of d to the physician. I find objectionable, since I not believe the physi should have to rely on source for final scien truth. There is also a p tical objection: Since physicians actually pense drugs, they sel see the package insert any event, I would m tain that the physic should know what dru wants and why without pending on the governm or the manufacturer to him.

Undoubtedly, physic are swamped by exces numbers of drugs in s therapeutic categories. I am well aware that m drugs within such ca gories could be elimina without any loss, or haps even some profit the practice of medic But, in my opinion, neit the FDA nor any o single group has the ex tise and the wisdom ne sary to determine the "drug of choice" in areas of medical practic

laker of Medicine

eth G. Kohlstaedt, M.D.,
Vice President,
ledical Research,
Lilly and Company

my opinion, it is not
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or private regulatory
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be labeled "the drug
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clinical experience is
nulated, new evidence
become available.
, it may be apparent

that the established products should not be so easily dismissed.

Variation in patient response to drugs constitutes one of the major obstacles to the determination of "drugs of choice." We are just beginning to open the door on pharmacogenetics, but it is evident that genetic differences cause wide variations in the way drugs are absorbed, metabolized, etc. This fact alone is sufficient to make unrealistic the idea that there is one drug in each class to be used for every human being.

The problem of determining relative drug efficacy is an extremely complicated one. Comparison with other drugs of the same class should not be a prerequisite for marketing a new substance. In some therapeutic areas, it may be difficult to make accurate comparisons. For example, in the treatment of infections it is not possible to conduct crossover studies. Recovery may be influenced by factors which cannot be controlled or measured, i.e., natural host resistance and virulence of infective agents. A drug's acceptability must often be judged on the basis of its own performance, and this may be limited to experience in a relatively small patient population. If the introduction of a new drug must await the adequate establishment of relative efficacy, the duration of clinical trial and extent of studies would be greatly prolonged, particularly for rare or unusual conditions. The availability of a new drug would be delayed. Many patients might suffer needlessly and lives might be lost.

Relative efficacy can best be established by experience in a general patient population through regular channels of clinical practice. The physician considers the patient as a whole, which means the patient often has multiple problems and drugs must be selected with this in mind. Hence, a "drug of choice" in an uncomplicated case may not be the best drug for a patient with associated problems. Publication of well-controlled studies in medical journals may provide comparative evidence; discussions at medical meetings, presentations at postgraduate courses, and the new audio-visual technology may bring evidence to physicians on comparative therapy. In a free medical marketplace, a drug that does not measure up will fall into disuse. For example, broad clinical experience has established vitamin B_{12} as the "drug of choice" for the treatment of primary pernicious anemia. No amount of advertising or promotional effort by the manufacturer could increase the use of liver extract for this anemia. However, a physician may wish to employ parenteral liver preparations for a special purpose.

In the field of surgery, peer review in the hospital has brought significant improvement in the use of new techniques and procedures. Something of this nature would be useful in the area of drug therapy. However, it should be developed by the medical profession itself and would necessitate, for its proper function, an improvement in the dissemination of reliable data on clinical pharmacology of drugs under consideration.

Ideally, information on the relative efficacy of drugs should be gathered and assessed by the physicians who actually administer the specific agents to a specific patient population. To do this, they will need even more information on the drugs they use — information that the pharmaceutical manufacturers must begin to provide if government regulation of *"drugs of choice"* is to be avoided.

Opinion & Dialogue

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SCIENTIFIC ARTICLES

Alpha-1 Antitrypsin Deficiency

JAMES J. LINN, M.D.*†

The first description of pulmonary emphysema was by Laennec in the early 19th century. It is a chronic obstructive pulmonary disease occurring mostly in males, usually in the 5th and 6th decades. There is often an antecedent history of chronic bronchitis. This is in sharp contrast to that found in the familial variety, first described in 1963 by Laurell and Eriksson.[2] In their series the patients had a family history of emphysema, which usually develops at an earlier age and with no previous recurrent pulmonary infections. Surprisingly, more females are afflicted with this form than in the ordinary variety. Also, there was a significant association between this form and a deficiency in serum alpha-1 globulin, or, more specifically, the alpha-1 antitrypsin.

Biochemical Properties of Alpha-1 Antitrypsin

The alpha-1 globulin is a glycoprotein with a molecular weight of 47,000.[3] It constitutes 2% of plasma proteins and 13% of carbohydrates. The total serum antitryptic activity is contained in 3 protein fractions, approximately 90% in the alpha-1 antitryptic globulin,[2] and the remaining 10% activity shared by the alpha-2 macroglobulin and an interalpha trypsin inhibitor.[4] The specific action of alpha-1 antitrypsin has not yet been completely elucidated; however, it inhibits such proteases as trypsin, chymotrypsin, elastase, and collagenase. Normally it is found in human serum, urine,[5] colostrom and lactating milk.[6] Its concentration increases with elevations of endogenous steroids and ACTH or corticosteroid administration,[7] surgical stress,[5] infections,[8] vaccinations, during pregnancy,[5,9] and with estrogen-progesterone preparations.[9]

Inheritance, Incidence, and Normal Values

In the original work done by Laurell and Eriksson[2] the trypsin inhibition followed a trimodal distribution, consistent with the theory that the deficiency state was inherited as an autosomal recessive trait. The highest values occurred in the serum of normal individuals, while the lowest values were found in young patients who had an unusual propensity for pulmonary obstructive disease. Intermediate levels were seen mostly in those who were heterozygous for the recessive gene.

It is estimated that anywhere from 1–2% of patients with chronic obstructive disease are homozygous for the anti-enzyme deficiency.[10] In a select group of patients Hepper and associates[11] found the incidence to be as high as 30%. These patients have serum values that are 10–15% that of normals, while their respective alpha-macroglobulins are increased 134%.[12,13] Those who are heterozygous show levels approximately 50–90% of normal.[12] There is no real correlation between the degree of deficit and the severity of the disease. It should be noted that nearly 10% of patients homozygous for the trait do not develop emphysema, which suggests that other factors may play a role in the disease.[14]

In a large series done by Makino et al[15] the geometric mean of the serum level of alpha-1 antitrypsin for 1,063 men between the ages of 15–40 was 310 (233–422) mg%, and the serum mean value for 1,390 females of the same age was 336 mg %.

Since the immunodiffusion test employed in this hospital lists its normal values as between 200–400 mg %, and with no differentiation between males and females, it was necessary to run a series of tests on normals to see if such a parameter affected the accepted values. Also, since heterozygous patients have values anywhere from 50–90% of normal, it becomes critical for a laboratory to establish its own range of normal values.

Materials and Methods

Serum was obtained and frozen for 7–10 days from 60 patients (28 females and 32 males) ranging in age from 16–71. This series was made up of outpatients, blood donors, and pre-operative patients scheduled for minor surgery. All denied current infections, pregnancy, oral contraceptives, or steroid therapy.

Quantitative determinations were performed on Partigen™* plates which contain a layer of agar gel into which anti-a1-antitrypsin (of rabbit origin) has been incorporated.' The agar layer contains 12 uniform cylindrical wells arranged around a central moisture chamber. Three dilutions of a standard solution of known alpha-1 antitrypsin were run on each plate. The plates were examined after 48 hours, and the diameter (in mm.) was plotted as the ordinate on semi-log paper, with the abscissa being the concentration in mg/100 ml.

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* Chief Resident in Pathology, Washington Hospital Center, Washington, D.C. when written.

† Awarded Honorable Mention at the Annual Philip Burka Award presentations, 1971.

Address reprint requests to Dr. Linn at the Department of Pathology, Washington Hospital Center, 110 Irving Street, N.W., Washington, D.C. 20010.

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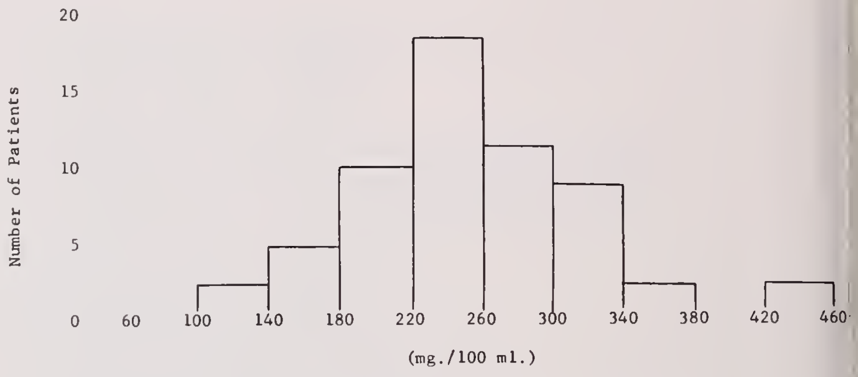

FIG. 1. Serum Concentration of A_1-Antitrypsin (All patients).

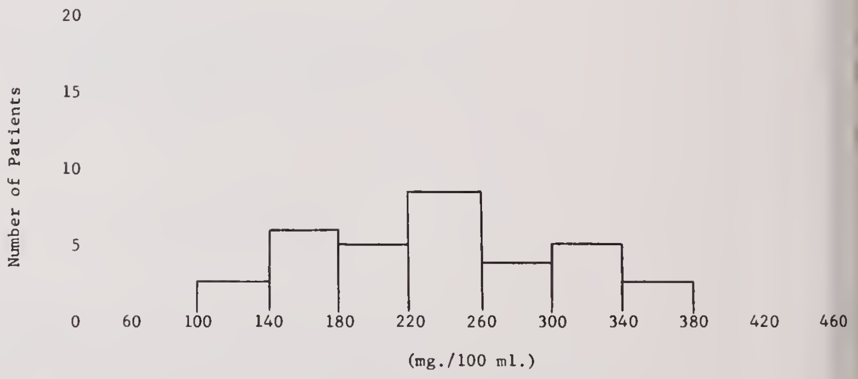

FIG. 2. Serum Concentration of A_1-Antitrypsin in Males.

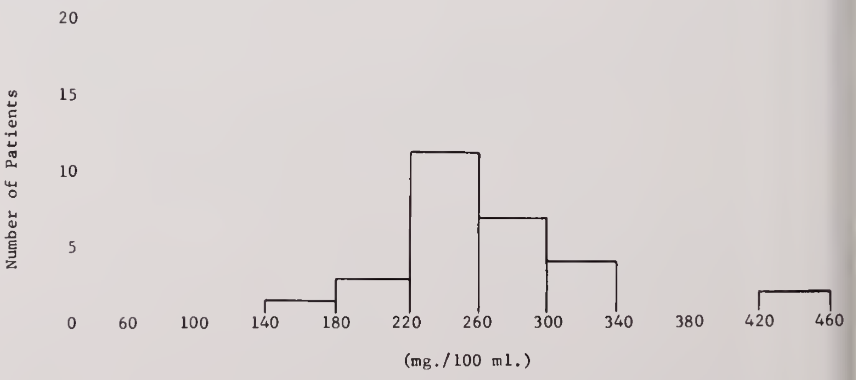

FIG. 3. Serum Concentration of A_1-Antitrypsin in Females.

Results

Figure 1 shows the distribution of values obtained in all patients. Figures 2 and 3 show the distribution of values in males and females. For all patients, the average value was 248.8 mg% with a range of 110–388 mg.% (± 2 s.d.). Apparently there is a difference between male and female patients. The average value for males was 231.5 mg% with a range of 91.4–371.6 mg% (± 2 s.d.), and for females 268.5 mg% with a range of 139.9–397 mg% (± 2 s.d.).

Other Associative Diseases

Subnormal but not deficient levels of alpha-1 antitrypsin have been observed in both nephrosis[16] and advanced liver disease.[17] Lower than normal values have also been seen in association with necrotizing angiitis and glomerulonephritis.[18] Lieberman[19] in his series found an unusual prevalence of peptic ulcer, although there was no statistical difference compared to a control series. Lopez et al[20] reported a case of asymptomatic lymphadenopathy in a male child with alpha-1 deficiency.

Discussion

As more cases are reported there appears to be a significant increase of pulmonary disease in heterozygous individuals.[21] Their disease is usually not as severe as that seen in homozygous patients, since in the former group they are capable of increasing their antitryptic activity in response to infections, thereby avoiding an unopposed tryptic activity.[14]

The most feasible hypothesis to explain the development of emphysema in antitrypsin-deficient patients is based on the observation that alpha-1 antitrypsin is capable of inhibition of the proteolytic enzymes released by leukocytes during inflammation, thereby preventing excessive tissue destruction.[22] Absence of this protein increases the susceptibility of pulmonary connective-tissue fibers to metabolic degradation with subsequent increase in the formation of extracellular fibrous protein.[23] This replacement of connective tissue fibers, while under continual tensile strength, leads to enlargement of the peripheral air sacs, a major factor in the developing process of emphysema.

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Indications for Penetrating Keratoplasty

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Corneal transplantation has become a feasible technique for the treatment of severe corneal disease only in the last 30 years. As the knowledge of corneal physiology has grown and as new techniques have evolved, the success rate of corneal surgery has increased. The last decade has seen remarkable strides in the development of new surgical techniques, the knowledge of pathological processes, and their control have grown apace. It is safe to say that keratoplasty, as it is optimally performed today, is a completely new operation which is more complex but carries with it an enormously improved prognosis for clear grafts. The outlook in previously bleak cases has been reversed, and conditions a few years ago thought to be contraindications to penetrating keratoplasty now have very favorable results. Because of this new and frequently misunderstood re-ordering of prognostic categories, a review of the current status is indicated.

Recent Advances

Until very recently, clouding of the corneal graft was thought to be synonymous with graft rejection. As our knowledge of corneal pathophysiology has grown, it is now realized that the "homograft reaction", i.e., graft rejection, is an infrequent occurrence. There are multiple other factors influencing the state of the donor endothelium and, hence, graft clarity. Poor tissue apposition, prolonged postoperative inflammation, vitreous touch, primary failure of the donor endothelium (lack of sufficient functional reserve), and release of lytic enzymes by diseased host tissue can cause graft clouding, either singularly or cumulatively. Efforts have been directed toward eliminating or controlling these factors. The introduction of anterior vitrectomy as a standard procedure in aphakic case has greatly decreased the incidence of vitreous touch.[1]

One of the major innovations has been the use of the operating microscope and new, extremely fine (22 micron) nylon suture. With the magnification afforded by the microscope, sutures can be placed more accurately, depth better assessed, and tissue handled less traumatically. Moreover, the 10-0 nylon suture excites much less inflammation than the older, larger silk sutures. This has resulted in better tissue apposition and less postoperative inflammation. These differences can be critical, especially in vascularized host tissue.

While these advances in surgical techniques represent a major contribution, postoperative care is equally important in graft prognosis. The judicious use of corticosteroids postoperatively minimizes inflammation, protecting the donor endothelium and effectively controls vascularization of the graft. Although excessive steroid dosage can inhibit wound healing, proper amounts do not significantly affect this, while controlling unwanted inflammation. Indeed, a vascularized host cornea was previously considered a contraindication to keratoplasty. This is no longer true; heavily vascularized host eyes have a very good outlook, properly managed. Careful postoperative management necessitates daily slitlamp examination and determination of intra-ocular pressure. With the earlier ambulation offered with the new suture material, hospitalization is reduced. The postoperative care period is lengthened because 10-0 nylon sutures can and should be left in place comfortably for 3-6 months before removal.

In those few eyes in which a bona fide "homograft reaction" occurs, the early institution of topical steroids can control the process effectively. Indeed, the prognosis for penetrating grafts has improved to the point where they have largely replaced the safer, but visually less satisfactory lamellar graft in this country.

Corneal Edema

The cornea exists in a precariously dehydrated state. This relative dryness of the corneal stroma is responsible for corneal clarity. Both the corneal epithelium and endothelium prevent imbibition of fluid by the stroma.[2] In addition, an active metabolic process within the corneal endothelium functions to maintain the stroma compact and deturgesced.[3] As a result, damage to the epithelium will result in moderate stromal swelling beneath the denuded area. This is usually readily reversible upon epithelium healing.

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Damage, however, to the endothelium results in much more severe stromal and/or epithelial edema because both the barrier and metabolic functions are compromised. Moreover, in the adult eye, epithelial repair does not readily occur.

Compromise of endothelial function can occur in a variety of ways. A gradual dystrophic process, increasing with advancing age, occurs in Fuch's endothelial-epithelial dystrophy. The diagnosis can be made by the slitlamp appearance of cornea guttata in combination with epithelial edema. In addition, surgical trauma to the endothelium can result in endothelial decompensation with corneal edema; the endothelium can be damaged by vitreous touch. Any or all of these factors may contribute to the ultimate development of corneal edema.

While stromal swelling results ultimately in scarring and moderate decrease in vision, accumulation of fluid within and beneath the corneal epithelium causes severe visual loss and frequently a painful bullous keratopathy. Recent experience with the use of the new soft contact lenses has shown that they relieve pain but do not consistently improve visual acuity significantly except in mild cases.[4,6] These mild cases of epithelial edema are frequently managed successfully with topical hypertonic agents, e.g., 5% sodium chloride, but eventually this will not suffice and visual acuity will drop. In patients with a visual acuity of 20/100 and below (provided there is not intractable glaucoma) and in whom vision is a primary consideration are candidates for penetrating keratoplasty.

Using current techniques and judicious postoperative management, a success rate of 85% in phakic cases, and 70% in aphakic cases is common.[7]

In patients with cataract and corneal edema a combined procedure carries with it an almost equally good success rate.[8] Sequential operations, i.e., keratoplasty followed by cataract extractions, are characterized by graft-clouding in 20%.[9] This is due to damage to the graft endothelium associated with the second operation.

Corneal Scars

The prognosis for clear grafts in corneas scarred by trauma or previous infection is very good. This is true without regard to the degree of vascularization of the host cornea. While the postoperative management differs in vascularized and non-vascularized host eyes, a clear graft rate of about 85 to 90% can be predicted.[10] A condition causing corneal scarring that needs qualification is interstitial keratitis. These eyes frequently have greatly increased postoperative inflammatory reaction.[11] Postoperative complications are more common. A recent report describes a success rate of 80%.[12]

There is one troublesome group within this area. Eyes with corneal scarring secondary to previous herpetic infection can be quite successfully grafted after the disease has been inactive for at least 6 months. Unfortunately, there is a tendency for reactivation of the herpetic infection to occur post-operatively in about one-third of patients.[13] This possibility can be minimized by prophylactic treatment with topical antiviral compounds but not entirely eliminated.

Corneal Dystrophies

If one eliminates Fuch's epithelial dystrophy, the remaining corneal dystrophies represent the most promising prognosis for penetrating keratoplasty. A success rate of 90% is common in cases of keratoconus, Groenouw's type I and II, lattice dystrophy, and other less common forms.[10] Results are most gratifying, and experience with long term follow-up exceeds 25 years in some cases. Grafts initially successful have remained clear over this period. Figure 1 is a postoperative photograph of the eye of an 11-year-old boy recently grafted on for severe keratoconus. The postoperative visual acuity is 20/20.

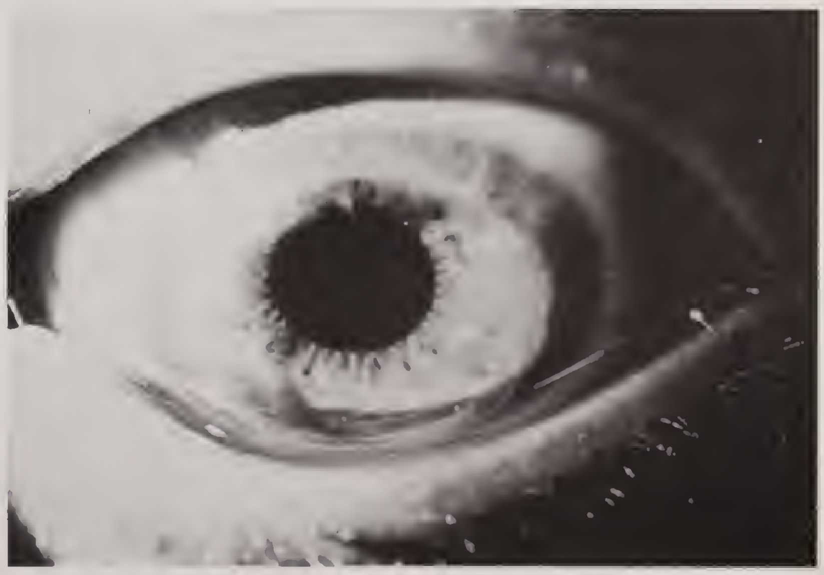

FIG. 1. Post-operative penetrating keratoplasty in an 11-year-old boy with keratoconus.

Dry-Eye Syndromes and Lid Problems

There are a number of ocular diseases characterized by a decrease in tear production. The most common is that of keratoconjunctivitis sicca, seen usually in menopausal women and frequently associated with a systemic disease, e.g., a collagen vascular disorder. In these cases there is a lymphocytic infiltration of the lacrimal glands with a decrease in the aqueous tear production.[14] While pain and photophobia are sometimes incapacitating, severe corneal desiccation with visual decrease is rare. In contrast, there are a number of less common diseases which are characterized by a decrease in mucin production by the conjunctival goblet cells, i.e., avitaminosis A, Stevens-Johnsons disease, and ocular pemphigoid.[15-18] In spite of an adequate volume of aqueous tears, the corneal surface is not wetted properly because of the lack of mucin, the principal factor in effecting wetting of the cornea. Severe corneal desiccation and opacification can occur. Related problems leading to similar desiccation occur in cases of 7th nerve paresis in which the lid cannot surface the cornea with mucin.

The treatment of these conditions has been hampered by the lack of an effective mucin substitute. Likewise, the prognosis for keratoplasty has been poor because of desiccation of the corneal graft. Research in our laboratories is currently under way to develop a long-lasting artificial tear preparation with muco-mimetic properties. In the meantime, the use of the new soft contact lenses in conjunction with artificial tear solutions offers a promising avenue in the postoperative management of these problems (table 1).

Keratoprostheses

Despite the major advances described above, there exists a small but tragic group of patients with diseases that do not respond well to conventional keratoplasty. These conditions include alkali burns of the eye, ocular rosacea, ocular pemphigoid, and Stevens-Johnson disease (the latter 2, for other reasons in addition to their decreased mucin production). A pathogenetic mechanism common to all these conditions is the abnormal elaboration of digestive enzymes by the diseased epithelium. These enzymes "melt" the corneal stroma in the postoperative period, frequently causing perforation and loss of the eye. In the past 5 years advanced designs for keratoprostheses or plastic corneas have offered some hope for salvation of these eyes.[19-21] Figure 2A is a pre-operative photograph of an eye which was severely injured with alkali 10 years ago. The visual acuity was light perception only. He had previously undergone 17 unsuccessful procedures. A (collar button) corneal prosthesis[20,21] was implanted on our Corneal Service 14 months age. Figure 2B is a recent photograph of this eye. The present visual acuity is 20/50, and the patient can read very fine newsprint. Only longer follow-up will reveal how long such devices will remain in place, but initial results are encouraging.

Miscellaneous

Other important new treatment modalities that have aided the handling of corneal disease include:

1. *Corneal adhesives in the treatment of corneal perforations.*[22,23] Cyanoacryate monomers, which are liquid, polymerize in the presence of free OH^- radicals to form a solid in seconds. When the monomer is applied on the small polyethylene disc to a corneal perforation of up to 2½mm in diameter, a solid tamponade of the hole is achieved (figure 3). The adhesive remains in place for a variable length of time while healing occurs beneath it. This technique has obviated the need for suturing the cornea or plugging the hole with preserved tissue in many cases and minimizes the scarring near the visual axis. The adhesive can be removed easily, once healing has occurred.

A second application of the adhesive is in the field of glued-on hard contact lenses. This field was investigated over the past several years, and glued-on lenses gave very good visual results, by providing a smooth optical surface in many cases of monocular aphakia (minimizing aneisokonia). The limiting factor was the duration of the adhesive

TABLE I: Prognosis for Penetrating Keratoplasty

Condition	Clear Grafts
Corneal edema (without intractable glaucoma)	
Phakic	85
Aphakic	70
Corneal scars	
Herpetic	85 (Recurrence of Herpes in approx. 1
Non-herpetic	90
Interstitial keratitis	80
Corneal dystrophies (excluding Fuch's)	90
Dry-eye conditions	Poor (figures unavailable)
Avitaminosis A	Candidates for keratoprosthesis
Stevens-Johnson disease	
Ocular pemphigoid	
Exposure keratitis	
Alkali burns	
Ocular rosacea	

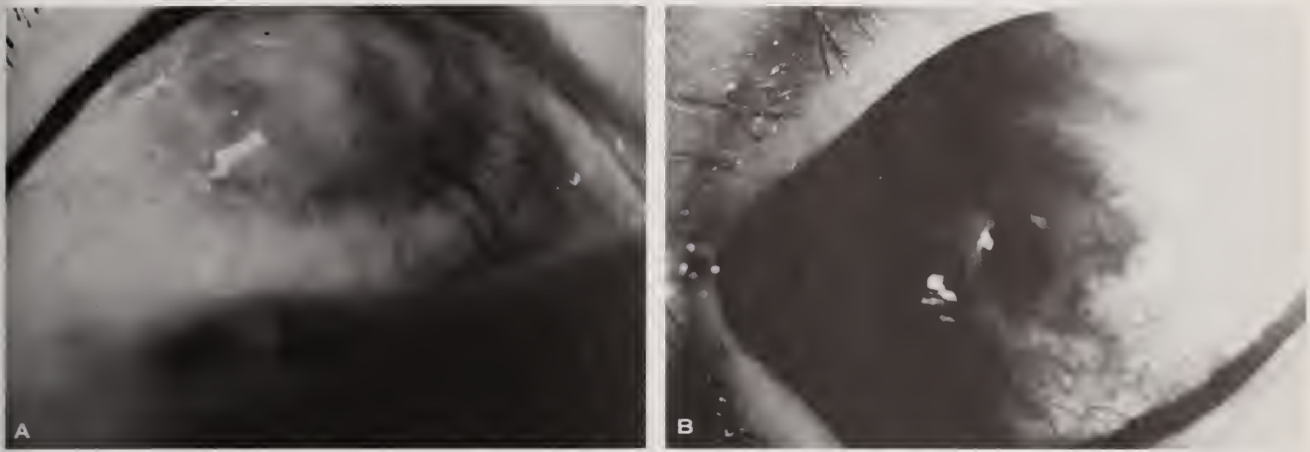

FIG. 2A. Pre-operative photograph of an eye severely injured with alkali 10 years previously. 9 months prior to this photograph a corneal prosthesis was implanted. 2B. This is a recent photograph of the eye. The visual acuity is 20/50.

bond which usually lasted only a period of months.[24] More recently, glued-on hard contact lenses are being used as a temporary treatment of severe alkali burns of the eye. The rationale is that the lens covering the cornea will prevent access of the digestive enzymes to corneal stroma, thus preventing corneal perforation which is a common occurrence in the first few months after injury. We are engaged in a collaborative study with several other institutions across the country in evaluating this treatment method.

2. *Anti-collagenolytic agents in the management of corneal ulcers.* Agents such as EDTA-Ca, cysteine and acetylcysteine which are inhibitors of collagenase, an enzyme liberated by diseased corneal and conjunctiva epithelium, may be useful in the treatment of severe corneal ulcers.[25,26] The dosage and relative effectiveness of these agents is not known. One of these (acetylcysteine) is commercially available as a bronchial mucolytic agent* and can be made up in normal saline as 10 or 20% solution. A novel approach is the use of this in conjunction with a soft contact lens. This may prolong the action of the acetylcysteine, but its ultimate value is yet to be determined.

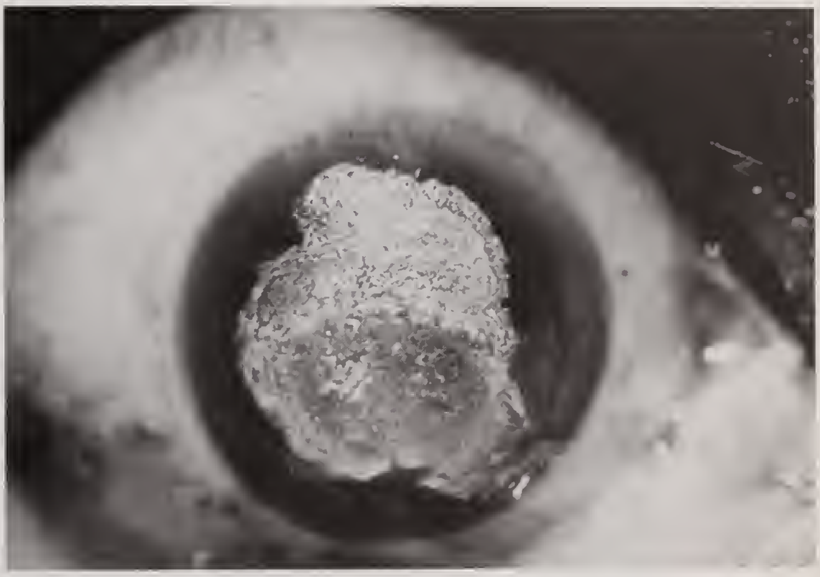

FIG. 3. A solid tamponade of the hole is achieved when the monomer is applied on the small polyethylene disc to a corneal perforation of up to 2-1/2 mm. in diameter.

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* Mucomyst.

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THE VIEW BOX

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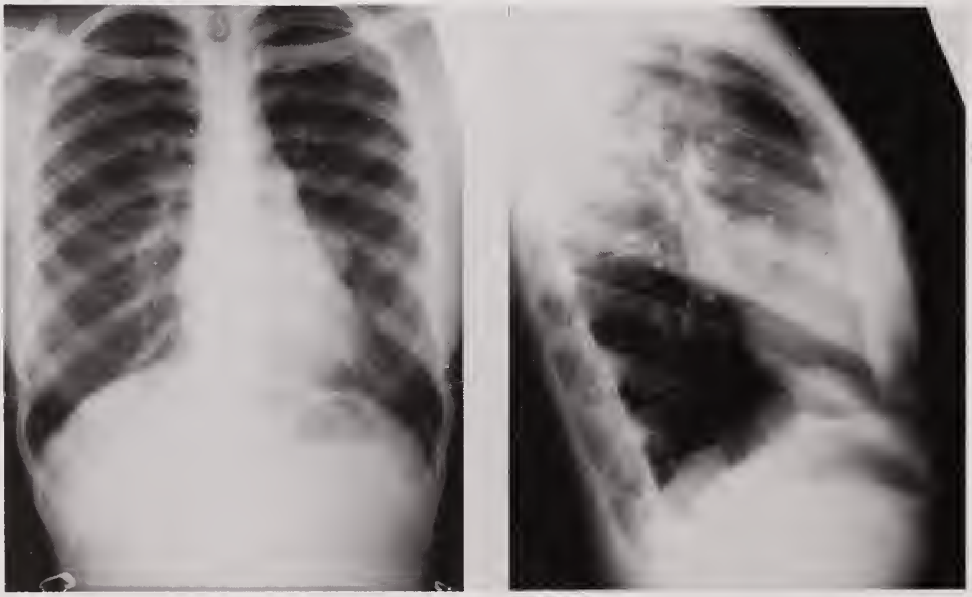

A 17-year-old was well until several days ago when she experienced a sudden cough with expectoration of a "mouthful" of blood. This was a single event. Physical examination and routine laboratory studies were normal.

(For diagnosis see page 363)

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Cardiovascular Manifestations of Myotonic Dystrophy

Report of a Case*

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Atrial flutter usually means serious organic heart disease[1,2], and persistence of this abnormal rhythm for extended periods of time is infrequent. This report concerns an exceptional case. Aside from an 8-year hiatus, atrial flutter was present for 24 years in a patient who was considered otherwise normal for 20 of these years. This peculiar case is reported to direct attention to heart disease, particularly atrial flutter, as the initial manifestation of myotonic dystrophy.

Report of Case

January 27, 1946, during a routine physical examination, the patient, then a 19-year-old seaman, was found to have an irregular heart rhythm. He was hospitalized at a Naval Hospital. An electrocardiogram showed "auricular flutter with irregular ventricular complexes". The patient reported no cardiac symptoms, and no abnormal physical findings were detected. Digitalis administration produced "4 to 1 atrioventricular block", but an attempt to restore normal rhythm with quinidine failed. During the ensuing 8 years he was repeatedly evaluated by cardiac consultants for the Veterans Administration. He complained of dyspnea and faintness with exertion but was regularly employed as a sales clerk. No abnormalities were present on examination. Electrocardiograms continued to show atrial flutter with variable atrioventricular block (figure 1). Digitalis did not alter the symptoms or the arrhythmia.

In 1954 the patient entered the Atlanta Veterans Administration Hospital, but a second attempt to revert the atrial flutter with quinidine failed. Quite unexpectedly in August 1954 he was found to be in normal sinus rhythm. This continued for 8 years. The patient complained of frequent palpitations and exertional faintness, and his examiners assumed that paroxysmal arrhythmia was occurring. In early 1962, established atrial flutter recurred and has persisted.

In January 1966 the patient complained of insidiously progressive difficulty in walking and exhibited a steppage gait. The neurologic findings were typical of myotonic dystrophy. Atrophy and weakness of the facial, sternocleidomastoid, and shoulder-girdle musculature were prominent. The intrinsic muscles of the hands were wasted, as were those of the calves. Myotonia was apparent, manifested as difficulty in the release of the hand-grip and in percussion myotonia

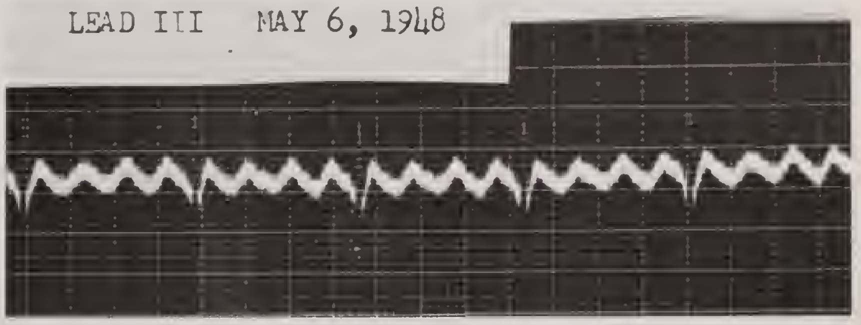

FIG. 1. Lead III of the electrocardiogram recorded in 1948 documents atrial flutter with 4 to 1 A-V conduction.

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of the thenar eminence. Testicular atrophy, frontal baldness, and punctate lenticular opacities completed the picture. Electromyograms were compatible with myotonia. Muscular dystrophy had not been known in the patient's family. No abnormal cardiovascular findings were present at this time, even though intraventricular conduction was now abnormal (figure 2). Chest X-ray films revealed moderate cardiac enlargement.

There have been no further changes in the cardiac status. Atrial flutter was still present in March 1970. Skeletal muscle weakness has been insidiously progressive.

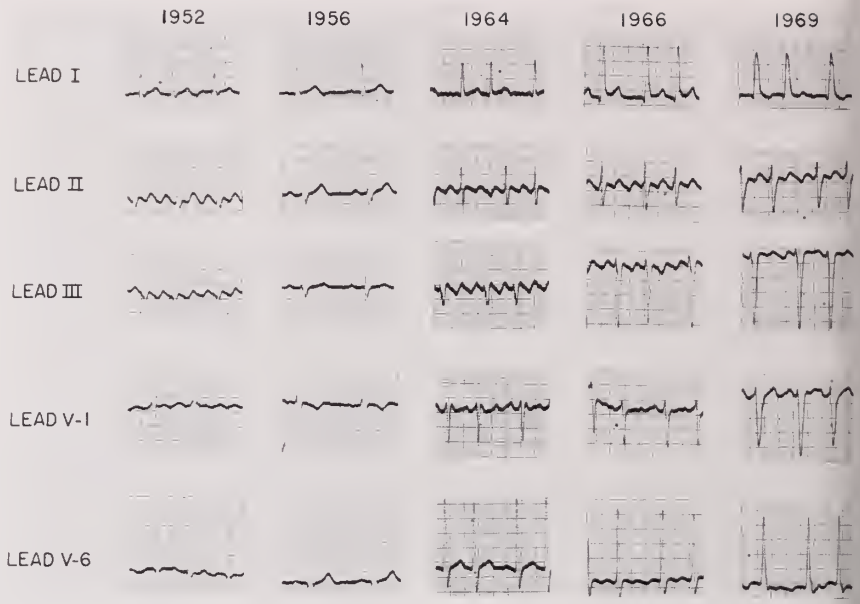

FIG. 2. Selected electrocardiographic leads from a 17-year span depict the development of the intraventricular conduction disturbance.

Discussion

As in the other forms of muscular dystrophy, widespread degeneration of skeletal muscles is found in myotonia dystrophica. Its hallmark is myotonia, inability quickly to release voluntary muscle contraction. Its propensity to produce weakness and atrophy of the distal musculature of the upper extremity is also distinctive. A characteristic facial expression resulting from involvement of the facial, masseter, and temporal muscle groups has been termed "myopathic facies". Footdrop, commonly an early symptom, reflects weakness of the anterior tibial and peroneal musculature. Frontal baldness, gonadal atrophy, and cataracts are frequent. The last is often seen in family members with little or no myopathy. As a rule, myotonia appears in the second or third decade and precedes weakness. If it is not severe, myotonia may be overlooked by the patient, particularly if he has not noted the disorder in his family. Skeletal muscle weakness may then be the initial complaint of the patient.[3]

Heart involvement by the degenerative process seems to be the rule. More than 300 reported cases of myotonic dystrophy were recently reviewed by Church.[4] Although symptoms referable to heart disease were not frequent, at least some alteration in the electrocardiogram was present in 85% of the 236 individuals in whom the recording was reported.

Prolongation of the PR interval is the most frequent abnormality. More severe atrioventricular conduction disturbances are unusual, but artificial pacing has been necessary.[5,6] Intraventricular conduction disturbances are also common. Left-axis deviation is very frequent, but bundle-branch block is also reported.[4]

Atrial arrhythmias are less common. Church[4] was able to discover 17 instances of atrial flutter and 4 of atrial fibrillation. Chronic atrial flutter was observed in 1 patient by Spurny and Wolf.[7] Electrocardiographic abnormalities, but not atrial flutter, antedated the diagnosis of myotonic dystrophy. Atrial flutter began after the recognition of the skeletal muscle disorder and persisted for 4 years. Another instance of chronic atrial flutter, quite similar to that of the patient herein reported, was studied at the National Institutes of Health.[8] Stokes-Adams attacks were recognized 17 years prior to the diagnosis of myotonic dystrophy.

Of particular interest to the physician performing routine physical or screening electrocardiographic examinations and to those seeing cardiac patients in consultation is the possibility that arrhythmias or conduction abnormalities may result from myotonic dystrophy before the patient or his physician has recognized the neuromuscular disability. A search for myotonia, muscular atrophy, frontal baldness, testicular atrophy, or cataracts may lead to the proper diagnosis in the perplexing patient who has unaccountable atrial arrhythmia, left-axis deviation, or disturbed A-V conduction.

Instances in which evidence of heart involvement preceded recognized muscular disease have not been common.[4] This may partly be due to the characteristic paucity of cardiovascular symptoms from such involvement, and to the physician's failure to be directed by the electrocardiographic abnormalities to a search for signs of the skeletal myopathy.

Summary

An unusual case of atrial flutter persistent, except for an 8-year-hiatus, for 24 years has been reported. Manifestations of myotonia dystrophica were recognized 20 years after the onset of the arrhythmia.

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The Nephrotic Syndrome in Hodgkin's Disease*

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The nephrotic syndrome in association with Hodgkin's disease has recently attracted considerable interest.[1-10] The pathogenesis of the renal lesions is not well understood, but the syndrome usually resolves when the Hodgkin's disease is brought under control.

We report here an additional patient whose renal lesion was studied with the electron microscope.

Report of Case

A 13-year-old white boy's illness was diagnosed as the nephrotic syndrome in November 1970. A thorough search for an etiology was unfruitful. Administration of prednisone therapy was begun. In January 1971 a biopsy specimen of an enlarged lymph node in the right cervical area revealed Hodgkin's disease of the mixed cellularity variety. Chest X-ray films revealed mediastinal widening. Diagnostic laparotomy with splenectomy was performed on February 23, 1971. No evidence of Hodgkin's disease was found in the abdomen. The patient's condition was classified clinically as II-A.[11]

The patient subsequently received irradiation to the cervical area, mediastinum, and retroperitoneal and iliac regions. During therapy the proteinuria began to clear up, and both the Hodgkin's disease and the nephrotic syndrome were in remission at the time when radiation therapy was completed.

Follow-up examination in December 1971 showed no evidence of either Hodgkin's disease or the nephrotic syndrome. A percutaneous kidney biopsy specimen was obtained on December 9, 1970 at the Medical University of South Carolina, Charleston. With light microscopy the interstitial tissue and vessels were seen to be normal. The glomeruli were minimally hypercellular because of mesangial proliferation and contained an increased amount of para-amino salicylic acid (PAS)-positive material within the mesangium. The capillary basement membrane was not thickened.

Electron microscopy of the specimen revealed occasional balls of electron-dense material in the lamina densa, the mesangial area, endothelial cells, foot processes, and capillary lumina (figure 1). The mesangial area was increased in size, predominantly because of the granular material and the mesangial processes. Foot-processes were focally fused.

An open-wedge biopsy specimen of the kidney was taken during laparotomy on February 23, 1971. At this time the patient had been receiving prednisone for about 2 months. By light microscopy the specimen showed only slight focal increase in mesangial cells. Electron microscopy again showed foot-process fusion of the glomerular epithelial cells (figure 2). The glomerular capillary endothelial cells were extensively swollen (figure 3), and the parietal capsular epithelial cells were hypertrophied (figure 4). Interstitial blood vessels showed focal endothelial swelling, perivascular edema, fibrosis, and occasional interstitial lymphocytes.

Comment

The renal pathology in this patient was similar to that of the 2 patients having Hodgkin's disease with the nephrotic syndrome described by Hamburger et al[4] and the patient of Ghosh and Muehrcke.[7] Kidney biopsy specimens of their patients showed lipoid nephrosis; electron microscopy showed fusion of the visceral epithelial foot-processes.

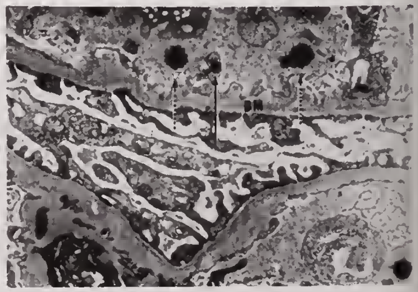

FIG. 1. Portion of glomerular capillary loop showing electron-dense material in an endothelial cell (solid arrow) and tubular lumen (broken arrow). BM—Basement membrane.

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The opinions or assertions contained herein are those of the authors and are not to be construed as official or as reflecting the views of the Bureau of Medicine and Surgery of the Navy Department or the naval service at large.

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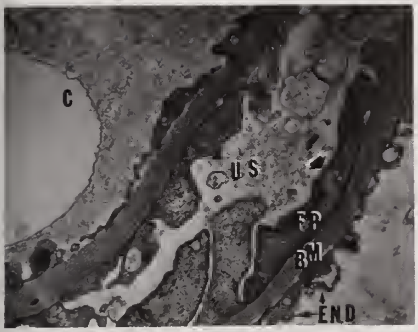

FIG. 2. Portion of glomerular capillary loop showing fusion of the foot processes (arrow) of the glomerular epithelial cells. C–capillary; US–urinary space; FP–foot process; END–endothelial cell.

One patient with this syndrome[6] was found by both light and electron microscopy to have true membranous glomerulonephritis. A second patient described by Miller[12] had typical membranous glomerulonephritis detected by light microscopy. Of the other patients reported having Hodgkin's disease associated with the nephrotic syndrome, 1 was found to have nonspecific glomerulonephritis[2] and 5 normal findings by light microscopy.[1,3,8]

The nephrotic syndrome has been found in association with malignant conditions other than Hodgkin's disease.[13-15] These reports state that the changes in the kidney were typical of membranous glomerulonephritis, except in the case of 1 patient with adenocarcinoma of the kidney and monocytic leukemia who had lobular glomerulonephritis and another patient with adenocarcinoma of the colon who had lipoid nephrosis.[13]

The explanation for the finding of the nephrotic syndrome in association with malignancy has been postulated to be the development of soluble immune complexes and their subsequent deposition on the glomerular basement membrane.[7,8] In support of this hypothesis is the finding of IgG immunoglobulin with immunofluorescent techniques on the glomerular basement membrane in 1 patient with bronchogenic carcinoma.[14] Further, it has been observed that the various types of immune complex disease seem to be commonly associated with certain varieties of malignancy, particularly lymphomas.[12]

NZB/B mice with malignant lymphoma develop a renal lesion similar to membranous glomerulonephritis.[16] Experimentally, a morphologic picture resembling membranous glomerulonephritis has been produced in the rat by the intravenous injection of an extract of the Walker 256 adenocarcinoma.[17]

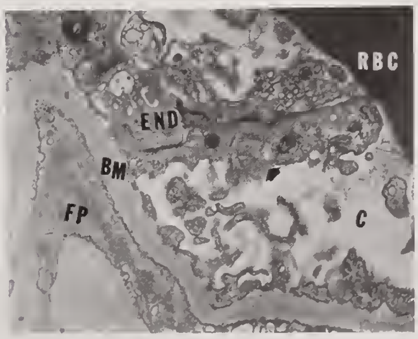

FIG. 3. Portion of glomerular capillary loop showing swelling of a glomerular capillary endothelial cell (arrow). RBC–red blood cell.

There is recent evidence for a tumor-associated antigen in Hodgkin's disease.[18] Whether such antigens play a role in the pathogenesis of the various immunological disorders associated with malignancy bears further study.

Until a firm association is determined between malignant disease and the nephrotic syndrome, a fortuitous occurrence of the 2 diseases cannot be dismissed. This may be true in the young person in whom the nephrotic syndrome develops more frequently.

Summary

The case of a patient who had the nephrotic syndrome in association with Hodgkin's disease has been presented. By light microscopy only slight mesangial proliferation was visualized, but by electron microscopy, foot-process fusion and hypertrophy of the glomerular capillary endothelial cells and of the parietal capsular epithelial cells were revealed. Electron-dense material was present within the glomeruli. The findings were consistent with the diagnosis of lipoid nephrosis and were similar to those seen in other patients with this syndrome.

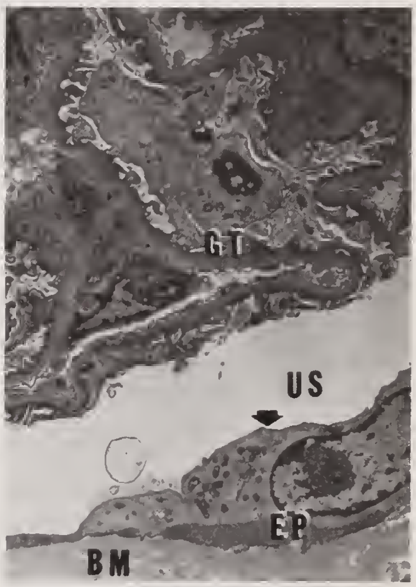

FIG. 4. Portion of glomerular capillary tuft (GT) with hypertrophy of a capsular epithelial cell (arrow). EP–epithelial cell.

Acknowledgment

The authors wish to express their appreciation to Miss Frances H. Atkinson for technical editing.

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Vitamin B_6 Dependent Convulsions Manifest in a Case of Down's Syndrome

A. MALIK MIRZA, M.D.* AND KADEKKAL VENUGOPALAN, M.D.†

Pyridoxine, pyridoxamine and pyridoxal are the 3 naturally occurring forms of Vitamin B_6. These are phosphorylated in the body to form pyridoxal-5-phosphate (PLP), which acts as a co-enzyme in a wide range of reactions. The key PLP dependent reaction, in causing the convulsions if defective, is thought to be the formation of gamma amino butyric acid from glutamic acid by glutamic acid decarboxylase,[2] the so called "GABA" shunt. Neonatal convulsions due to pyridoxine dependency were first described by Hunt et al.[3] Since that time a number of other reports[2,4,5] in the siblings have appeared relating the cause as genetically determined impairment of Vitamin B_6 co-enzyme activity. The neuro-chemical and clinical aspects of pyridoxine have been discussed in detail[1,8] and reviewed by Rosenburg.[9] We have had a case of pyridoxine dependency in association with Trisomy 21.

Report of Case

The patient was a female infant born to a 13-year-old Negro, primigravida after 4 hours of labor. The membranes ruptured 30 minutes before delivery. The mother received 100 mg of Demerol 1 hour prepartum. The infant with vertex presentation was delivered normally, Apgar 9 at 1 minute. Examination in the delivery suite revealed a normal baby weighing 2.7 Kg and 47 cm long. At 7 hours of age she was noticed to be cyanosed and having jerky movements of the extremities. She was immediately transferred to the sick-baby unit and placed in isolette with oxygen. She was cyanotic with a bulging fontanelle, mongoloid facies, a distended abdomen, and exaggerated reflexes. Phenobarbital 6 mg intramuscularly and 8 mg within 30 minutes was unable to control the convulsions. Intravenous fluids started with 10% glucose and 10% calcium gluconate 7 ml were given twice without any effect on the convulsions. During this period blood chemistries spinal fluid examination were normal.

The infant was kept on 10% glucose intravenous maintenance fluid. At 16 hours of age 50 mg of Pyridoxine was given intramuscularly. The patient became relaxed within 5 minutes. Hyperthermia was noticed after the Pyridoxine injection. The temperature rose to 39.4°C, necessitating sponging. The baby had an uneventful recovery and was placed on Enfamil feedings. Repeat subdural taps were negative. The head circumference remained unchanged. The patient was discharged on the 10th day on Enfamil and Trivisol, 0.6 ml daily.

On the 15th day of life and 5th day after discharge the patient was readmitted with convulsions. She had tonic and clonic generalized convulsions with facial twitchings. She emptied her bladder once but did not pass stools. She had circumoral cyanosis and exaggerated reflexes. These convulsions recurred every 5 to 10 minutes and lasted 15 to 20 seconds. Intravenous glucose, 2 ml of calcium gluconate and intramuscular phenobarbital were tried without control of the convulsions.

Blood glucose was 243mg%, BUN 13mg%, sodium 139mEq/L, potassium 6.5mEq/L, chloride 101mEq/L, CO2 11mEq/L, calcium 8.7mg%, and phosphorus 6.7mg%. Blood, stool, nasopharyngeal and spinal fluid cultures were negative. A urinalysis was negative. An EEG on 3 occasions was reported to show nonspecific activity without gross abnormality. A karyotype showed mosaicism, trisomy 21. A tryptophan load test showed abnormal xanthurenic acid excretion after a loading dose of 100 mg per Kg of tryptophan (22.4 mg in 24-hour urine.)[6,7]

Course

The patient was given 100 mg of pyridoxine intramuscularly with immediate control of the convulsions.

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She was then placed on oral pyridoxine, 10 mg daily. She again manifested hyperthermia following pyridoxine injection. The temperature rose to 40.5°C but was brought down by sponging. The infant was noticed to be irritable while off pyridoxine during the xanthurenic acid test.

Enfamil and cereals were given for 15 days. The formula was changed to evaporated milk for 2 days and convulsions recurred. These were controlled by 15 mg pyridoxine by the intramuscular route. Hyperpyrexia was not present at this time.

The patient has since been maintained on oral pyridoxine, 12.5 mg daily. The mother, due to her immaturity, could not take the child home. The grandparents did not show any interest in the child. Hence the patient was placed in an orphanage. She has remained well.

Comment

The cause of pyridoxine dependency is not known. It is believed that convulsions are due to genetically-induced disturbed utilization of pyridoxine. The exact site of the action of pyridoxine is still not clear. The key enzyme involved is thought to be glutamic acid decarboxylase. The prompt response of the convulsions to pyridoxine is clear indication of no abnormality in conversion of the co-enzyme to pyridoxal-5-phosphate. Oxygen consumption is diminished in pyridoxine-dependent seizures, and gamma amino butyric acid is important in cerebral oxidative metabolism. This indirect evidence has been used by Scriver[2] to postulate the abnormality in one of the co-enzyme binding sites of the apo-enzyme glutamic acid decarboxylase.

The occurrence of this disease in families indicates some genetic defect. However, there are no specific studies to implicate any specific chromosome responsible for the defect.

The absence of pyridoxine dependency in cases of Trisomy 21 and its presence in this particular case makes it unlikely that Group 21 chromosomes are involved in the control of pyridoxine metabolism. However, we can speculate that the patient developed Trisomy 21 due to pyridoxine dependency. This may have to be evaluated in the later studies.

The case presented here typifies some of the characteristics of pyridoxine dependency. The patient had 3 convulsive episodes in the neonatal period specifically controlled by pyridoxine. However, there are some features for consideration. The mother, in this case, did not take excessive amounts of pyridoxine during pregnancy, contrary to Hunt's reports.[3] She had 1 prenatal visit quite late in pregnancy and took the usual multivitamin and iron preparations. She noticed excessive movements of the baby, but again it was her first pregnancy. The reports of fetal distress,[5] i.e., meconium staining, cyanosis, retracting, and grunting were absent in this case.

The usual response of hypothermia following pyridoxine administration was not seen in this case, rather hyperthermia manifested itself. This was dose-related since it occurred during the early 2 convulsive episodes controlled by high doses of pyridoxine and not in the third, which was controlled by a relatively small dose. The exact mechanism of hypo- or hyperthermia is not known.

It is important to diagnose this condition early in order to institute proper treatment and prevent mental retardation.

Acknowledgements

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Tuberculoid Leprosy: A Neurologic Case Report

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Leprosy remains an infrequently encountered disease within the continental United States. Estimates range between 2,000 and 4,000 cases, but because of increasing world travel and the inherent difficulty of early establishment of the diagnosis, these estimates may be unduly conservative.

Within a 10-year period from July 1, 1955 to June 30, 1965, 362 first admissions were recorded at the Public Health Service Hospital at Carville, Louisiana, an average of less than 40 patients per year.[1] Not all persons in the United States with leprosy require hospitalization, and such statistics are not truly representative of the incidence of leprosy and must remain largely speculative.

Report of Case

A 21-year-old Thai national had a 2-month history of pain and paresthesias in the left elbow and weakness of the left hand. The pain came frequently at night, particularly with the patient lying on the involved side. The weakness was initially mild, and increased until he became unable to hold objects or perform movements requiring sustained finger effort in the left hand. No trauma had occurred to the neck, shoulder or limb. The history was negative for infections or neurologic disease.

Examination revealed pronounced atrophy and weakness of all interossei, the musculature of the hypothenar eminence, as well as weakness of the flexor carpi ulnaris in the left hand. Decreased pain and temperature sensation were noted over the dorsum of the 4th and 5th fingers, and a cord-like thickening was palpated in the upper arm. This decreased in bulk and thickness at the olecranon process. There was no other peripheral nerve involvement, and the rest of the neurologic examination was negative.

Nerve-conduction velocity studies showed absence of response on stimulation of the left ulnar nerve at the wrist or elbow with the median and radial responses remaining normal. Electromyography demonstrated complete absence of voluntary motor units in the hypothenar musculature and the first dorsal interosseous. These studies were consistent with degeneration of the left ulnar nerve with degeneration and atrophy of the musculature supplied by this nerve.

An open-nerve biopsy revealed a thickened nerve deep to the area between the biceps and triceps on the medial aspect of the left arm. Nerve stimulation at surgery failed to elicit evidence of nerve conduction to the appropriate muscles. Upon dissection of the nerve covering, a yellow caseous material was found at various portions along the nerve. Biopsies of these areas confirmed the clinical impression of "granulomas consistent with leprosy".

Aggressive treatment was begun with Dapsone.

Discussion

In more than 50% of the patients diagnosed as having leprosy, neurologic involvement was the presenting symptom. Usually anesthesia or paresthesias are the presenting complaints. Although seen with other neuropathies, they are frequently the precursors of leprosy. They may progress to intense neuralgia and episodes of lightning-like pains in the affected limb or merely remain as an area of localized hyperalgesia of the skin. In all cases of anesthesia or paresthesia, leprosy remains a realistic possibility, particularly in patients from Texas, Louisiana or Florida, States with the highest rate of leprosy.[3]

A history of prior contact with leprosy is often unobtainable. Half of the leprosy patients born in the continental United States have no knowledge of the source of their infection.

Among the classes of patients in whom leprosy is most likely to appear are veterans, especially those with prior service in the Far East, foreign service personnel, whose travels take them to Mexico, the Philippines, the Pacific Islands, Central and South America, Asia, Hawaii and even Puerto Rico; and medical personnel from the Philippines and Pacific Islands who have emigrated to the United States for employment.

The importance of early diagnosis of this eminently treatable disease can hardly be overstated. Early treatment limits disability and deformity as well as eliminating a potential source of infection from the community.

Unfortunately, early diagnosis is more often the exception than the

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rule. An average delay exists of 17.4 months from the time a physician is first consulted to the establishment of the diagnosis. When this is added to an average delay of 14.5 months from the onset of symptoms until the patient seeks medical attention, the total lapse of time prior to initiation of appropriate treatment approaches 3 years.

The differential diagnosis includes disorders marked by anesthesia without accompanying loss of mobility, and the patient is able to freely use the anesthetic limb. Such a pattern can occur in diabetic peripheral neuropathy, congenital indifference to pain, hereditary sensory radicular neuropathy, and syringomyelia. Leprosy is characterized by preserved deep-tendon reflexes despite cutaneous sensory loss of a distinctive pattern involving the "cooler" parts of the body and the presence, as in this case, of enlarged non-tender peripheral nerves, especially the ulnar, medial, and common peroneal. In addition to these usual sites, other mixed nerve trunks may be similarly affected-the radial nerve in its groove, the popliteal nerve deep in its fossa, and the dorsalis pedis. Occasionally gross enlargments of small cutaneous nerves are found close to developing leprous lesions of the skin in such unlikely places as the knee or the elbow, the so-called "Medusa's head" of radially disposed nerves.[6] The great auricular nerve may be enlarged when the skin of the sternocleidonstoid is stretched taut. In India this is recognized as a stigma of the disease.[7]

Regarding the sensory deficit in leprosy, a great variability exists in both the order in which the sensory modalities are affected and in the degree of this impairment.[8] The inability to demonstrate any sensory deficit impairment does not rule out the diagnosis if other suggestive evidence of the disease can be demonstrated, such as enlarged nerves and pigmentary changes. Amyloidosis, hypertrophic interstitial polyneuritis, and leprosy are the 3 entities in which enlarged nerves can be palpated on physical examination.[9] Definitive diagnosis can be made by biopsy, but the clinical picture is sufficiently distinctive to allow a fairly definite diagnosis on clinical grounds alone.

The patient is usually a male with a history of residence in the areas endemic for leprosy. His complaints involve numbness, cramps, weakness, and pain or tenderness. Because leprosy is basically a disease of the skin, nerves and mucous membranes, other symptoms may include pruritus, hoarseness, nasal congestion. If the infection is localised, as in tuberculoid leprosy, small anesthetic abnormally pigmented areas may be discovered within the area of nerve dysfunction which is localized. Lepromin skin-testing may be positive in such cases. In lepromatous leprosy many peripheral nerves show dysfunction with widespread symmetric involvement. Careful search for enlarged nerves narrows the diagnostic range still further. When discovered, biopsy of the involved nerve is diagnostic.

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Hypothermia Associated with Methadone Intoxication*

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Methadone was synthesized in Germany and introduced into medical practice in 1946 as an analgesic agent;[1] currently, however, it is used in maintenance programs for the treatment of opiate addiction.[2] Although hypothermia occurs with both methadone and opiate administration, it is usually mild, with the body temperature depressed from 0.1° to 1.7°C.[3,4] This report describes a state of profound hypothermia associated with methadone overdosage in an individual in whom tolerance for the drug was lost during a period of imprisonment.

Report of Case

The patient is a 22-year-old man who was a heroin addict for 3 years until 6 months prior to admission when he discontinued the drug and entered a methadone maintenance treatment program. One month prior to admission, while taking 150 mg of methadone per day, he was arrested and incarcerated for 30 days. Mild withdrawal symptoms were experienced when methadone was discontinued during his confinement. The day following his release, 14 hours prior to admission to the hospital, he ingested his previous customary dose of methadone, 150 mg. After a brief period of euphoria he went to sleep in his heated apartment. The next morning his wife was unable to arouse him and observed pink frothy material around his nose and mouth. He was brought to the Emergency Room where he was unresponsive and cyanotic. The blood pressure was 60 mm Hg by palpation, the radial pulse 60 beats per minute and irregular, the respiratory rate 4 per minute, and the temperature 33°C rectally.† Needle tracks were observed along the forearms, and the pupils were markedly constricted but equal in size. Fine inspiratory rales were heard anteriorly and posteriorly throughout both lung fields. The apical heart rate was 80 beats per minute and irregular. No cardiac murmurs were audible. No focal neurological deficits were demonstrable.

Initial arterial blood gases revealed: pO_2 58 mm Hg; O_2 saturation 79%; pH 7.12; pCO_2 89 mm Hg, and CO_2 content 29.5 mEq/L. Nalorphine injected intravenously produced only a transient improvement in respiration. Positive pressure ventilation was achieved with nasotracheal intubation and the Bennett MA-1 respirator. Repeat arterial blood gases, while 100% oxygen was being inspired, revealed: pO_2 79 mm Hg; O_2 saturation 92%; pH 7.21; pCO_2 76 mm Hg, and CO_2 content 31 mEq/L. The initial electrocardiogram (ECG) showed artrial fibrillation with a ventricular response of 80 beats per minute and J waves (Fig. 1 top). The central venous pressure (CVP) measured 29 cm. H_2O, and the chest x-ray was compatible with pulmonary edema.

Three episodes of ventricular fibrillation which were successfully defibrillated with ultimate reversion to atrial fibrillation occurred during the first 2 hours of hospitalization. An intravenous infusion of isoproterenol was titrated to maintain the systolic blood pressure at 80 mm Hg.

Four hours after admission the cardiac rhythm spontaneously converted to sinus. Although the rectal temperature had risen to 35°C, a small J wave was still present in the ECG. Body temperature was maintained at 35°C rectally with a hypothermic blanket.

Following the intravenous administration of ethacrynic acid and digoxin the clinical condition improved. Within 12 hours after admission the CVP decreased to 8–12 cm H_2O. The arterial blood gases while 80% oxygen was being inspired were: pO_2 150 mm Hg; O_2 saturation 98.9%; pH 7.46; pCO_2 46 mm Hg; CO_2 content 33 mEq/L.

The hypothermic blanket was discontinued 24 hours after admission. When the rectal temperature rose to 37.5° C the J waves disappeared. Thirty-six hours after admission, blood pressure and ventilation were satisfactory and stable without aids. Evidence of gradual lessening of the pulmonary edema with clearing after 72 hours was noted radiographically.

Hemoglobin, hematocrit, white blood cell count, sodium, potassium,

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† The lower limit of the IVAC 820 thermometer, IVAC Corp., San Diego, California, is 33°C.

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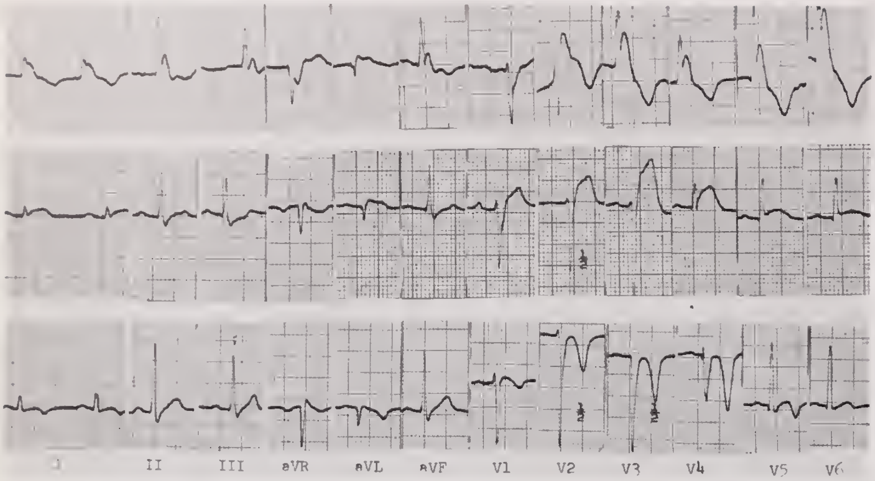

FIG. 1. (top) Admission ECG revealing atrial fibrillation and J waves; (middle) ECG at 48 hours, revealing acute anterior injury; (bottom) ECG at 19 days, compatible with an anterior myocardial infarction.

chloride, bilirubin, blood urea nitrogen, alkaline phosphatase, serum glutamic-pyruvic transaminase determinations, and urinalysis were normal. The serum glutamic oxaloacetic transaminase (SGOT) was 45 units (normal: less than 50 units), and the lactic acid dehydrogenase (LDH) was 250 units (normal: less than 200 units) on admission; 48 hours later, the SGOT rose to 540 units and the LDH to 1100 units, and then both gradually returned to normal.

An ECG recorded 48 hours after admission revealed sinus rhythm with ST-segment elevation in Leads I, AVL, and V_2 through V_6, consistent with acute anterior injury (Fig. I middle). The ECG 19 days later was compatible with a recent anterior myocardial infarction (Fig. 1 bottom) and was confirmed by a vectorcardiogram which revealed absence of anterior forces.

The patient was discharged in good condition without medication.

Comment

Treatment of opiate addiction in a methadone maintenance program is initiated with a small dosage of methadone which is gradually increased to 80-120 mg per day.[2] Upon withdrawal of the drug, tolerance is lost, and the previous maintenance dose may be toxic. An oral dose of 70 mg in a non-tolerant individual can be lethal.[5] The toxic effects of methadone are similar to those of morphine and heroin and include vomiting, bradycardia, respiratory depression, pulmonary edema, and mild hypothermia.[1, 3-6]

The degree of hypothermia measured in this patient, as indicated by the rectal temperature, at most 33°C, and the presence of J waves on the ECG, is more severe than that associated with the administration of opiates. With the induction of hypothermia the initial cardiac rhythm becomes sinus bradycardia and is then followed by atrial fibrillation; if the temperature is decreased further, ventricular fibrillation occurs. A deflection, the J-wave, occurring during the terminal portion of the QRS complex is characteristic of severe hypothermia[7] and usually appears when the rectal temperature is less than 30°C.[8] When present, the J wave is a premonitory sign of ventricular fibrillation,[8,9] as in this case. Although atrial fibrillation, possibly due to heightened vagal tone, has been reported with heroin overdose,[10] the presence of J-waves in our patient's ECG would indicate that the cardiac arrhythmias were more likely due to the severe hypothermia.

With increasing numbers of narcotic addicts receiving treatment in methadone programs, the potential lethal effects of the drug, especially in patients who have lost their tolerance, and in non-tolerant family members of patients, should be noted. Patients recently imprisoned, or otherwise maintained drug-free, should be regarded as non-tolerant, and if treatment is resumed, small dosages of methadone rather than previous maintenance dosages should be administered.

Summary

Profound hypothermia developed in a former heroin addict after the ingestion of 150 mg of methadone 4 weeks after withdrawal from its regular administration. He responded to ventilatory and circulatory support and was discharged from the hospital without sequelae. This case emphasizes the potential lethal side-effects which may occur following methadone overdose.

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THE VIEW BOX

(from page 350)

DIAGNOSIS: Bronchial adenoma–carcinoid type.

These lesions are epithelial tumors that arise from ducts of mucous glands of the bronchial tree. Histologically there are three forms:

1. Carcinoid type, 85-95% – located almost always beyond level of carina
2. Cylindroid type, 5-15% – in proximal bronchi or trachea
3. Muco-epidermoid type – Very rare and very malignant

This lesion is of low grade malignancy. In a small number there is local invasion. In 10% of the cases metastases occur either in local lymph nodes or distant sites. Malignant changes are higher in cylindromas.

Because the bronchial adenoma is very vascular, hemoptysis is a common symptom and occurs in about 50%. Other symptoms are dependent upon the degree of obstruction that is produced with atelectasis and secondary infections. Occasionally the carcinoid type gives rise to the carcinoid syndrome.

ROENTGEN FINDINGS:

1. A nondescript, well circumscribed, often lobulated nodule is seen in the central or, less often, in the peripheral lung fields. Calcification is rare.
2. Partial, or more often, complete atelectasis where the adenoma itself often is not seen.

Literature reference on request.

Confidentiality and Privileged Communications: The Psychotherapeutic Relationship Revisited

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The private self has thoughts, feelings, fantasies and information whose revelation would embarass and possibly harm the individual. Legal scholars debate whether the law recognizes the fundamental right to keep personal data of this type inaccessible to the public eye. Beyond the concept of the private self, there are levels of personal disclosure which the individual may sanction.[1] Considering privacy as the first level, the next level is that of confidentiality—the limited communication of certain information to another person for a certain purpose. Given an implied (or express) promise not to divulge this information, the right to confidentiality exists. Clearly, this is one of the basic tenets of the physician-patient relationship. The third level is that of privileged communication—the legal protection which shields the confidant from contempt or other court sanctions when he refuses to divulge the confidence in a legal proceeding. Such silencing requires statutory enactment. As with all 3 levels, this right belongs to the relator of information and may be waived by him only. It exists to encourage him, when seeking help, to be honest with the helping agent.

Statutes have been enacted to protect the right to privileged communication between various professionals and their clients—attorney-client, priest-penitent, physician-patient, and, more recently, psychotherapist-patient. The purpose of this paper is to explore the ramifications and development of this latter privilege.

To date 10 states have enacted a statute which establishes a privilege specifically for psychiatrist-patient communications. They are: California, Connecticut, New Hampshire, Florida, Georgia, Illinois, Kentucky, Maryland, Massachussetts and Tennessee. In the absence of such a statute, a medical physician-patient statute may be construed to apply to the psychiatric fact-situation. All but Alabama, Delaware, South Carolina, Texas, and Vermont have such a privilege. Some states have a psychologist statute which protects communications between patient and licensed psychologist. The statutes and their judicial interpretation vary widely in terms of who is considered a professional, what proceedings are involved, and which exceptions to privilege are defined. This situation makes it mandatory that physicians be aware of the statutes in the jurisdiction in which they practice.

Ninety per cent of all cases which invoke privilege are in 1 of 3 categories: 1) actions on policies of life, accident or health insurance, 2) actions for damages for personal injury or wrongful death, 3) testamentary actions in which the mental competency of the deceased is the principal issue.[2] In terms of psychiatric privilege, the information desired may be factual, predictive or diagnostic; it may also concern the very existence of the psychiatric relationship.

In reviewing the history of privileged communication, we find that at common law the only recognized privilege was that of attorney and client. There are not a few instances of physicians being prosecuted for attempting to protect patient's confidences. The first statutory intervention came in 1828 when New York granted privilege for physicians. Other states were slow to follow suit, but by 1960 more than 30 states had a physician-patient privilege, although none differentiated psychiatrists from other physicians.

Eventually, some states came to recognize licensed psychologists as entitled to a similar privilege. In some of these states, which lacked a psychiatrist-patient statute, the result was ironic—psychologists were entitled to a more liberal privilege than psychiatrists, who were categorized under the physician-patient statute. Finally, in the mid-1960's Georgia and Connecticut led the way in passing psychiatrist-patient privilege statutes. These have been the model for other states.

Let us turn to the theoretical arguments for psychiatric privilege.

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Psychotherapists of all types have been enthusiastic in their sponsorship and support of privilege. The most obvious reason is their conviction that privacy and absolute trust are critical to the psychotherapeutic interaction. As Freud said, "The whole undertaking becomes labor lost if a single concession is made to secrecy".[3] Iconoclast Thomas Szasz, who has proposed a system of autonomous psychotherapy, agrees that privacy is essential. "The role of the analyst is never to transmit information to third parties." he argues, because all information is derived from the patient anyway, and if a third party wants information he should get it from the individual.[4]

The argument for psychiatric privilege derives from the distinctiveness of therapy as opposed to medical consultation. The patient in analysis must learn to free associate and to break down resistances to dealing with unconscious threatening thoughts and feelings. To revoke secrecy after encouraging such risk-taking is to threaten all future interactions. To quote one author, "It would be as if a physician invited a patient to undress to be examined and then allowed the law to see him naked and arrest him for exhibiting himself."[5]

The effective core of all psychotherapies is the interpersonal relationship regardless of the specific method of treatment. Freudians, Sullivanians, Jungians, Behaviorists, Rogerians and Existentialists, to mention but a few, all agree that the privacy of the relationship must be protected.

The other major impetus to secrecy derives from the peculiar value placed on psychiatric treatment in our society. In spite of our efforts to educate people to the contrary, the general public still regards such consultation with distrust and suspicion. The danger, then, is that such negative sanctions that disclosure may have upon the relationship will destroy the present interaction and threaten treatment for the future, both on an individual and a societal basis.

The profession also argues that psychiatric testimony by the therapist is very seldom useful to the court. The patient, confronted with litigation, may instinctively seek the psychiatrist as a friend, projecting that his testimony will be a useful addition, but this is often untrue.

The therapist will be forced to condense hours, often years, of information and insight into mere minutes on the witness stand. His data are entirely subjective anyway and open to entirely different construction by another "expert." Cross-examination will distort his version of the story. The jury will misinterpret. Even his diagnosis may have been a label chosen to satisfy administrative or insurance form needs.

Proponents of psychiatric privilege point out that the court has access to other forms of professional evaluations, which can provide effective testimony without violating the therapeutic situation. The court may appoint a psychiatrist to examine and report about a litigant. Such an evaluation often can be made with greater objectivity than one by a psychiatrist who has established a therapeutic relationship with the patient. This is both more useful to the court and protective of the interaction.

Lack of privilege can open the door to the legal fishing expedition of discovery, and extended interrogation under deposition can be a vindictive search.

Arguing against privilege, the legal profession is committed to the principle that full disclosure amidst the rigors of the adversary system somehow will attain a just determination of the right decision. The extreme of the argument is a sort of legal paranoia, the suspicion that the content of privileged communication is precisely the information which would be most damaging and reprehensible if revealed, especially since one could find experts willing to support any diagnosis. There is no question that the law recognizes in other areas the public policy value of disclosure. It has legislated such disclosure in the areas which it judges of concern: gunshot or stab wounds, narcotics, abortion, child abuse.[6]

In states with medical privilege the trend has been toward greater restriction. One measure which considerably curtails the scope of privilege is to make it contingent upon judicial discretion, as was suggested by the 1938 American Bar Association Committee on Improvement in the Law of Evidence.[7] Proponents of privilege insist that the discretionary preliminary emasculates the statute beyond significance. Their most serious objection is that the predictive and normative value of the statute is lost if it is discretionary; patients cannot choose the depth of their revelation based on privilege since they cannot know what communications will be disclosed.

In 1969 the Committee on Rules of Practice and Procedure of the American Bar Association published their preliminary draft of proper rules of evidence, including their model psychiatric privilege provision. When the ABA responded to the proposed rules they rejected its report. Instead, they proposed the abolition of all privileges for physicians and psychiatrists in the interest of disclosure and pursuit of truth.

Are patients deterred in actuality by the absence of such privilege? Opponents of privilege argue convincingly that there is no lack of patients in a non-privilege state, nor do physicians report secretive, reticent patients in these jurisdictions. On the other hand, the act of consulting the psychiatrist is often considerably more difficult than consulting another physician. Hollingshed and Redlich[8] pointed out:

"Such concerns may be even more prevalent among persons in lower socio-economic groups, who are most difficult to reach in psychiatric treatment. Such persons tend to be more than ordinarily suspicious of authority figures and of the prospect that their disclosure will be used to their disadvantage."

In considering statutes which provide privilege, we have seen that when there is no specific psychiatric privilege this area is usually covered by the general physician-patient statute. In these states the protected communication must always be related to treatment. Other than that, different jurisdictions vary as to whether they include information acquired by observation, information gained from other physicians, or whether privilege applies to special legislative committee hearings. Since the relationship, demands and priorities of the psychiatric encounter are different from those of a general medical nature, they deserve special consideration and protection.

Some states which lack statutory privilege have a sort of informal extra-legal privilege which is extended to physicians and psychiatrists through the courtesy of the courts and the local bar. When Jonas Rappeport[9] analyzed the experiences of possible psychiatric witnesses in Maryland he found that, "What actually happened is that either the lawyer decided not to call the psychiatrist, the unacceptable questions were not asked, the judge let them go unanswered, or else the doctor 'lost records' or 'forgot' certain communications."

In fact, the reality of the psychiatrist and the courts is very different from what this entire discussion of privilege may imply. For the theory, we look at the position statement of the APA on Confidentiality and Privileged Communication with Specific Reference to Psychiatric Patients (1968).[10] This paper urges that a psychiatrist should never reveal information unless under court order. Even if the consent of the patient is granted he should protect the welfare of his patient as he sees it. If privilege is waived he must reveal all information. He must resist the subpoena until all efforts are exhausted. But in reality the psychiatrist, faced with an official, documented subpoena, often tends to assume that this is an order without recourse. A hospital is often still more susceptible to this sort of quasi-legal pressure, since the medical records librarian may send out privileged information in response to such an order without ever informing the patient or physician involved. Frequently psychiatrists allow their patients to think there is statutory protection for confidentiality when there is none; when there is a privilege law, they may violate it because they are unaware of its existence.[11]

The Group for the Advancement of Psychiatry proposed in 1960 its model legislation for psychiatric privilege—that this privilege be the same as the attorney-client.[12]

A. S. Goldstein and J. Katz[13] then published their critique of the proposal and found it flawed in its simplicity. It failed to note the problem areas and distinctions which the state statutes later took into account. The GAP model had such obvious flaws that GAP itself rescinded its proposal and supported instead the model statute drafted at Yale for the state of Connecticut 2 years later.

Of the statutes in effect in various states, there are 3 basic types, as exemplified by Connecticut, Massachussetts and California. One section creates the privilege, the second defines the terms, and the third defines exclusionary conditions. The differences and variations in the statutes reflect varying philosophies about some very basic problems.

First, there is the controversial issue of which professionals (or paraprofessionals perhaps) are included in the privilege. Probation officers, welfare workers, social agencies, and child-guidance counselors may all develop confidential interpersonal relationships which may deserve a psychiatric privilege. Different states protect different categories.

A closely related issue is the problem of third parties who may have access to private communications or confidential data. This may be a nurse's aide or another member of a therapeutic group of family members involved in the patient's treatment. None of the states has satisfactorily resolved this question, but California protects family members.

The question of the existence of therapy itself is another problem area. The mere fact that a psychiatrist was consulted may itself be confidential information which the patient would wish to be privileged.

The majority of courts have held that the State-appointed psychiatrist does not get privilege under the medical statutes, but if in addition he treats the patient, he does. But how does one draw the line? What sort of questioning is therapeutic instead of exploratory?

The court has indicated its abhorrence for confession under narcosis and under subtle pressure from examining psychiatrists. Even without statutory provisions the courts have held that a hidden eavesdropper to such an examination (particularly a policeman) creates privilege. And if the examination is made for the adversary party and the patient is never so informed, privilege is also erected. A similar situation exists in the Army, whereby unless a defendant is warned of his rights under article 31, Universal Code of Military Justice, evidence gathered in such examination is inadmissible by reason of possible self-incrimination.

The law equates disclosure of confidential communications to third parties with waiver of privilege. But a serious concern is whether any psychiatric patient has the ability to waive privilege. Is he aware of the consequences of disclosure and of the material which it will bring to light? Is he ever able to freely opt for publication of this information or do circumstances always force the issue? In terms of actual practice the patient may waive privilege simply by his failure to object to subpoena or deposition of privileged material.

There is the danger that a self-destructive individual may use his therapist's testimony to punish or sacrifice himself. Another basic concern is that the patient who seeks help in court from his therapist may be unleashing information which he never dreamed would be detrimental to his cause. The patient must be protected against this.

Does the therapist who discloses the confidential communication risk implicating himself as an accessory to crime? The question is more subtle when one considers all the non-heinous offenses which people usually "confess" to their psychiatrists. In many states fornication, homosexuality, and other sexual practices are illegal; these are usually matters for psychiatric perusal. The therapist must be cognizant of this risk.

The confidence of a plan to commit a future crime or tort may be an exception to privilege in some states. If treatment and rehabilitation of criminals is the goal of society, is this goal served by thwarting the possibility of treatment without incarceration for such individuals? What about the cathartic value of ventilating such threats as a positive measure in itself? It is frequently the job of the psychiatrist to help the patient deal with his destructive impulses to prevent his acting on them. The psychiatrist must point this out to the court and use privilege accordingly.

There are a number of other problems. Government agencies or prospective employers may require a person to sign a waiver of privilege for investigatory purposes. These may not be consented to voluntarily, and the psychiatrist must decide how much, if any, information he will reveal, and whether this will help or harm his patient. If he refuses to reveal information on ethical grounds, the investigator may assume that he is hiding some important facts.

Institution records also pose a difficulty. Some of the States extend privilege to hospital and clinic records, but not all. Thus, care must be exercised to record only those facts the publication of which would not be detrimental to the patient.

Another question is whether the privilege extends to communication with deceased patients. This issue frequently arises in relation to questions of testamentary capacity. It is difficult to avoid postmortem disclosures when the will is contested, but some states extend privilege to such communications.

The various statutes also extend privilege to different types of proceedings, civil, criminal or administrative, and cover different types of communications, such as research studies, diagnostic single-visit evaluations, and letters and phone calls.

Finally, the patient-litigant exception to privilege often exists when the patient waives privilege by bringing an action to court. The rationale is that by bringing into issue a set of facts, the patient has waived privilege and indicated a willingness to discuss that issue. But is privilege an all-or-nothing affair? One court said, "The patient is thus not obligated to sacrifice all privilege to seek redress for a specific mental or emotional injury."[14] But not all courts will extend this kind of understanding.

Joseph E. Lifschutz, a San Francisco psychiatrist, went to jail when he refused to obey a court order which waived the privilege of his patient under the patient-litigant exception. The California Supreme Court granted a writ of habeas corpus but upheld the exception and forced him to testify.[15,16]

Lifschutz wanted to test the statute and to overturn the exception in favor of an extended privilege. He argued on constitutional grounds that the proper practice of psychiatry demands absolute confidence; hence by abridging this, the state was invading his privacy, his patient's, and his own economic interest as a practicing psychiatrist. The patient had not said whether he wanted his doctor to testify, and Lifschutz wanted to claim this privilege for him, since waiver here was automatic, not voluntary. Moreover, the California priest-penitent privilege, which does not exclude litigants, was claimed to be denial of equal protection to psychiatrists.

The court was quite sympathetic with the concerns of the psychiatrist but voiced its own concern that to grant absolute privacy would close access to psychiatric information even when the patient chose disclosure. The court admonished trial courts to control compulsory disclosure with all available measures, such as in chambers conferences. It ruled, however, that the right of privilege is the patient's, not the doctor's.

Finally, there is a special area of concern in considering psychiatric privilege. This is the use of the computer in the mental health field, both for information storage and retrieval and for test evaluation. It has 'unlidded' a Pandora's Box of controversy about the issues of privacy, confidentiality, and privileged communications.

People like Senator Sam Ervin, Jr., Chairman of the Senate Subcommittee on Constitutional Rights, fear that although mass-data banks will effectuate new degrees of economy and efficiency, this would be accompanied by an invasion of privacy and possible infringement of the First Amendment rights to free speech and assembly.[17]

For health data systems, when the information is received in a confidential manner under the control of medical professionals, the primary legal issues are confidentiality and privilege. William J. Curran,[1] who studied this issue for Massachussetts, concluded that once admitted to an agency record, information protected by a personal right to privacy is no longer protected. This is because the Supreme Court has yet to recognize a corporate or agency right to privacy.

The matter is further confused by the lack of statutory consistency within the states. One forensic medicine specialist has pointed out that a national health data network cannot be implemented until national standards for privileged information are updated.[18]

Electronic safeguards are available to physically protect the information from unauthorized scrutiny. The Computers in Military Psychiatry Project at Walter Reed General Hospital aptly points out that the issue is not automation, but rather confidentiality.[19] Man creates these systems, and man can abuse them. Enlightened legislation and ethical codes must be contoured to ensure privilege of mental health information regardless of the manner in which it is handled.

In conclusion, the field of psychiatric privilege is replete with contrary theories and statutory variances. The psychiatrist's best preparation is to understand both the fundamental issues involved with privilege and the particular legal determinations in the jurisdiction where he practices. The profile of psychiatry is changing, as is the legal attitude toward privileged communication. As we become more involved in the areas of community and social psychiatry, using newer modalities such as therapeutic groups and para-professionals, new demands both for dissemination of information and for protection of information will be placed upon us. In the face of all those changes, the basic need to protect our patient's right to privacy still exists. It behooves us to constantly protect this right and to work to aid the legal community in developing enlightened attitude and policies in this area.

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The Stokes-Adams Syndrome*

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The Stokes-Adams syndrome is defined as an abrupt transient loss of consciousness due to a sudden but pronounced decrease in the cardiac output, which is caused by a sudden change in the heart rate or rhythm. This definition does not include vasovagal syncope or epilepsy, although patients with Stokes-Adams syncope may have seizures during periods of cerebral ischemia. Although partial or complete heart block is usually present during asymptomatic periods, many arrhythmias may produce syncopal episodes with or without the presence of previously established complete heart block. "Arrhythmia-induced syncope" is a more specific term and includes the primary cause of the decreased cerebral blood flow.

Clinical Features

The clinical manifestations of arrhythmia-induced syncope depend upon the duration and type of underlying arrhythmia as well as the status of the cerebral circulation. Symptoms of arrhythmia-induced syncope vary from slight faintness to loss of consciousness, with or without convulsions.

Characteristically, during the attack there is an initial pallor. Following resumption of the normal circulation there is usually a facial flush due to reactive hyperemia. The absence of an aura tends to separate seizures occurring during Stokes-Adams syncope from seizures of primary cerebral origin. Stokes-Adams seizures usually commence and terminate abruptly. The patient may resume a previous conversation or activity without being aware of the pause produced by the period of arrhythmia-induced cerebral ischemia. A slow or very rapid pulse during the period of unconsciousness points toward the correct diagnosis. Electrocardiographic monitoring during a syncopal episode demonstrates the responsible rhythm and makes appropriate therapy possible.

Etiology

Since most patients with arrhythmia-induced syncope have some impairment of atrioventricular (A-V) conduction either during or between attacks, the etiologies of Stokes-Adams syncope are often the causes of complete heart block.

Structural lesions of the heart. Approximately 7% of the cases of complete heart block in adults are congenital in etiology, with or without associated cardiac defects. Syncopal episodes complicating congenital heart block are uncommon but do occur and may necessitate pacemaker insertion.

Myocarditis of various causes may involve the conduction system, resulting in complete heart block and syncopal episodes. Diphtheria has long been known to be associated with conduction defects, particularly complete heart block.

Heart block, occasionally with Stokes-Adams syncope, has been reported during the course of connective-tissue disease and in association with degenerative skeletal muscle and nervous system disorders. In valvular heart disease valve calcification or endocarditis may involve the conduction system and produce incomplete or complete heart block.

Acute myocardial infarction is complicated by complete heart block in approximately 2 to 7% of cases. Complete heart block complicating inferior wall myocardial infarction usually proves to be transient, and restoration of normal conduction occurs within 2 to 3 weeks. The conduction defect involves the A-V junctional tissue. When an anterior wall infarction is the cause of complete heart block the mortality rate is extremely high, even with pacemaker insertion. In this situation the complete heart block is frequently due to extensive myocardial necrosis involving the right bundle and the 2 divisions of the left bundle (trifasicular block) rather than to a conduction defect in the A-V junction.

In a considerable number of patients no etiology can be established for complete heart block. Careful pathological examination has shown that many such patients with heart block do not have extensive coronary artery disease but have areas of fibrosis involving the conduction system distal to the common bundle (trifasicular block), either alone or in association with scattered areas of fibrosis throughout the myocardium. In elderly people this has been attributed to "sclerosis of the left side of the cardiac skeleton" which presumably results from wear and tear due to the repeated pull of the contracting left ventricular musculature.

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Primary and metastatic neoplastic disease, metabolic disease, and infiltrative disorders of the myocardium may produce heart block. Complete heart block is a potential risk during corrective heart surgery, particularly during repair of ventricular septal defects. Surgical heart block frequently reverts to sinus rhythm within 3 to 4 weeks following operation. If it continues beyond this period a permanent pacemaker is usually inserted. Non-penetrating chest injury is an occasional cause of complete heart block.

Heart block has been reported in association with Reiter's syndrome, amyloidosis, Pagets disease, and sarcoidosis. The myocardium is involved in approximately 20% of autopsy-proven cases of sarcoidosis. Conduction disturbances, Stokes-Adams syncope, and sudden death are known to occur in sarcoidosis and this entity is an important consideration in the differential diagnosis of acquired complete heart block in the young adult.

Electrolyte disorders. Potassium is the most important electrolyte in relation to A-V conduction. Isolated instances of advanced A-V block have occurred in patients during or following the administration of large doses of potassium salts. Hyperkalemia markedly decreases the ventricular rate in pre-existing heart block.

Acidosis depresses the ventricular pacemaker in complete heart block and may precipitate Stokes-Adams syncope. Alkalosis, hypokalemia, and hypernatremia increase A-V conduction and may reverse heart block. However, alkalosis and hypokalemia may increase ventricular ectopic activity, leading to ventricular tachycardia or fibrillation.

Toxic effect of drugs. Digitalis is the drug most frequently responsible for producing complete heart block. Syncopal episodes are uncommon during heart block due to digitalis toxicity because the ectopic pacemaker frequently originates in A-V junctional tissue and the ventricular rate is faster than in complete heart block of other etiology. The supraventricular rhythm is these patients is often atrial fibrillation.

The antiarrhythmic agents, quinidine and procaineamide, depress the conduction system as well as pacemaker rhythmicity and are contraindicated in complete heart block. Diphenylhydantoin and Lidocaine may also depress the ectopic pacemaker, and are therefore contraindicated in the presence of high degree A-V block. However, any of these antiarrhythmic drugs may be safely employed to suppress premature contractions in the presence of heart block if an adequately functioning electrical pacemaker has been inserted.

During the past few years markedly improved cardiac monitoring has more precisely elucidated the underlying electrocardiographic mechanisms producing Stokes-Adams syncope. Moreover, the rapid advances in electrical pacemakers have contributed significantly to the prevention and treatment of arrhythmia-induced syncope.

Electrocardiographic Mechanisms

At one time ventricular standstill was thought to be the sole mechanism responsible for Stokes-Adams syncope and is still considered the most frequent underlying rhythm disturbance. However, it is now established that Stokes-Adams attacks may also be due to extreme bradycardia or to a variety of tachyarrhythmias, particularly ventricular tachycardia and ventricular fibrillation. Continuous monitoring has demonstrated that several electrocardiographic mechanisms may produce syncope in the same patient.

The arrhythmias responsible for Stokes-Adams syncope may be divided into 7 groups:

1. Sudden interruption of atrioventricular impulse transmission causing transient asystole. When the cardiac rhythm changes from a sinus mechanism or incomplete A-V block to complete heart block, a period of asystole often occurs before the junctional or ventricular pacemaker assumes rhythmicity at its inherent rate. This "warm-up period" varies from 10 to 90 seconds and is termed the "pre-automatic pause."

2. Atrial standstill with failure of the junctional pacemaker resulting in ventricular asystole. When sinoatrial node impulse formation ceases and the A-V junctional tissue fails to assume rhythmicity, ventricular asystole results. This mechanism for Stokes-Adams syncope may occur in patients with inferior-wall myocardial ischemia and may be influenced by vagal hyperactivity. This is an uncommon mechanism of arrhythmia-induced syncope.

3. Asystole in the presence of established heart block. This arrhythmia may result from a shift in the pacemaker below the area of non-conduction to still a lower focus resulting in a period of asystole resembling the "pre-automatic pause."

4. Paroxysmal ventricular tachycardia or fibrillation in the presence of complete heart block. A slow heart rate during complete heart block predisposes to rapid impulse formation from ectopic foci. Either ventricular tachycardia or fibrillation may then ensue with syncope resulting.

5. Paroxysmal ventricular tachycardia or fibrillation during normal A-V conduction. These arrhythmias are most frequently observed in patients with acute myocardial infarction but have also caused syncopal episodes in patients with apparently normal hearts.

6. Supraventricular arrhythmias. Supraventricular tachycardias and bradycardias associated with syncopal episodes have been demonstrated by continuous electrocardiographic moni-

toring in many patients with the Stokes-Adams syndrome. Frequently the sinus bradycardia predisposes to episodic supraventricular tachycardia ("bradycardia-tachycardia syndrome"). In patients with coronary artery disease tachycardia increases myocardial oxygen demands and decreases left coronary artery diastolic blood flow, often decreasing cardiac output despite the increase in heart rate. Syncopal episodes may result.

Sinus bradycardia, sinoatrial block and sinoatrial arrest may cause syncope in patients with heart disease who are unable to increase stroke volume sufficiently to maintain adequate cerebral blood flow.

7. Combined forms. Uncommonly, paroxysmal tachyarrhythmias may be followed by a period of asystole due to a delay in automaticity of pacemakers which have been suppressed during the tachycardia.

The recognition that different electrocardiographic mechanisms may produce syncope in the same patient on different occasions is important in therapy.

Treatment

The aim of therapy for arrhythmia-induced syncope is threefold: (1) prompt restoration of the circulation during cardiac arrest, (2) restoration of an intrinsic cardiac rhythm adequate to maintain cerebral blood flow, and (3) prevention of recurrent episodes.

Medical treatment. The medical therapy of complete heart block includes the correction of potential contributing factors such as acidosis and hyperkalemia. The sympathomimetic drugs, which include parenteral epinephrine, oral ephedrine and isoproterenol by either route of administration, are primarily indicated when ventricular asystole or bradycardia occurs in complete heart block and intracardiac pacing is unavailable. These agents act by increasing A-V conduction, increasing the rate of the ventricular pacemaker, and shifting the lower pacemaker to a higher focus in the common bundle or A-V junctional tissue. During an attack of arrhythmia-induced syncope due to bradycardia, isoproterenol should be given by intravenous infusion during ECG monitoring so that its administration can be rapidly terminated if ventricular irritability results.

The vagalytic agent atropine may increase the ventricular rate in complete heart block complicating a recent inferior wall myocardial infarction. Atropine is often successful in the treatment and prevention of Stokes-Adams syncope due to sinus bradycardia, sinoatrial block and bradycardia-tachycardia syndrome.

Steroids through their anti-inflammatory and hypokalemic effects have been occasionally successful in improving A-V conduction in patients with complete heart block due to myocarditis or acute myocardial infarction.

The trisodium salt of EDTA (ethylenediamine tetra-acetic acid), a calcium chelating agent, has been used in the treatment of complete heart block due to digitalis intoxication when electrical pacing is unavailable.

Pacemaker therapy. Because of the unpredictable, potentially fatal nature of Stokes-Adams attacks and the inconsistent results and frequent complications with drug therapy, electrical pacing has become the treatment of choice when syncope occurs in patients with complete heart block.

The general indications for pacemaker insertion include (1) complete heart block associated with congestive heart failure, (2) complete heart block with Stokes-Adams syncope, (3) complete heart block following acute anterior or inferior wall myocardial infarction, (4) partial A-V block (second degree block) complicating anterior-wall myocardial infarction, and (5) post-surgical complete heart block.

Recent reports have demonstrated the feasibility of suppressing episodes of ventricular tachycardia and fibrillation in patients with normal A-V conduction by pacing the atrium or the ventricle at a rate faster than that present between episodes of ventricular tachyarrhythmia. Electrical pacing has been employed successfully in combination with propranolol and cardiac sympathectomy in the treatment of otherwise unresponsive ventricular arrhythmias. Rapid atrial pacing has been used successfully in the treatment of supraventricular tachycardias including atrial flutter and paroxysmal atrial tachycardia.

In patients with syncopal attacks due to ventricular tachycardia or ventricular fibrillation complicating complete heart block the emergency insertion of a ventricular pacemaker is strongly indicated. A ventricular pacemaker is the only means available for the long-term prevention of ventricular tachyarrhythmias in patients with heart block. If transient ventricular asystole complicates complete heart block a ventricular pacemaker is also indicated. A single Stokes-Adams attack in a patient with complete heart block is sufficient reason for pacemaker insertion.

The Syndrome of Midsystolic Extra Sound and Late Systolic Murmur*

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Mid-late systolic clicks and the associated late systolic murmurs are frequently encountered in clinical practice. Concepts concerning the generation of these auscultatory phenomena have undergone rapid evolution in the last 10 years. Traditionally, these sounds were considered to be benign extracardiac events generated by the motion of pleural-pericardial adhesions. However, it is now established that they are related to mild mitral regurgitation and emanate from tension on a large ballooning, redundant posterior mitral valve leaflet. Moreover, in a substantial proportion of cases there is a strong familial association of mid-late systolic extra sounds and late systolic murmurs indicating a congenital etiology for the valvular abnormality.

Auscultation

The mid-late systolic extra sound or click is a high pitched transient, usually best heard at the apex but often radiating widely. The most salient feature is its extreme variability. The sound may vary with position and/or respiration in an unpredictable fashion. Its quality, intensity, and position in the cardiac cycle can vary from moment to moment without provocation. In certain patients the sound is appreciated only in the standing positions, while in others, only in the left lateral or the supine position. Thus, careful auscultation will be necessary to detect its presence. Frequently, there is an associated systolic murmur of mitral regurgitation. In most instances the murmur will be confined to late systole and follow the systolic click. However, pansystolic and midsystolic murmurs also occur with the systolic extra sound and also indicate mitral regurgitation. The murmur also displays some variability but to a lesser degree than the sound. It may be high-pitched and is frequently located at the apex with poor radiation. The murmur, as well as the sound, may be difficult to hear, and again careful auscultation is necessary.

Cineangiocardiography

The cineangiocardiograms in the majority of patients with either systolic extra sounds or late systolic murmurs show a peculiar retroversion of the posterior mitral leaflet during mid to late systole. The valve leaflet balloons backward like a sail caught in a strong wind and fills with contrast medium. The systolic extra sound occurs at the height of the excursion of this redundant leaflet. The floppy valve can easily be seen in either oblique position. Frequently, there is a mild degree of mitral regurgitation, usually confined to late systole.

Pathologic Studies

There have been few postmortem examinations reported in patients with these auscultatory phenomena. Barlow reported a man who died suddenly and who had an enlarged redundant posterior leaflet with normal chordae tendineae. We have examined the the valves of 2 patients with midsystolic extra sounds, late systolic murmurs, and abnormal cineangiocardiograms. The findings were similar in both patients. The anterior leaflet area was 2-4 times larger than normal, while that of the posterior leaflet was 4-6 times greater than expected. Both patients had extensive deposition of a sulfated mucopolysaccharide in the media of the valve leaflets while the chordae tendineae, as well as the coronary arteries, were normal.

Clinical Syndrome

The majority of patients with either midsystolic extrasounds or late systolic murmurs are asymptomatic, and the auscultatory phenomena are found incidentally. Two major symptom-complexes merit discussion. Many of the patients complain of chest pain. The pain is substernal and "crushing." It is brought on by exercise but may occur at rest. It lasts minutes to hours and is poorly responsive to nitroglycerin. The pain has been attributed to angina pectoris, coronary insufficiency, myocardial infarction, and non-coronary chest pain, depending on which components are most prominent in a particular patient. Coronary arteriograms have been normal in the patients studied. The bouts of prolonged chest pain rarely are accompanied by enzyme rises. In a recent study of young females with chest pain, positive exercise tests and normal coronary arteries, 4% had the midsystolic click-late systolic murmur syndrome. The exact etiology of the chest pain is currently obscure.

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These patients frequently complain of palpitations. Several types of arrhythmia have been documented, including premature ventricular contractions, exercise-induced ventricular tachycardia, paroxysmal atrial fibrillation, and paroxysmal atrial tachycardia. Etiology is not entirely clear. Sudden death may occur in patients with the syndrome and among their first degree relatives.

Electrocardiograms

Electrocardiograms are frequently abnormal in patients with late systolic murmurs and/or mid-late systolic extrasounds. The abnormalities consist of T-wave flattening and inversion in leads II, III, aVf $V_{4\text{-}6}$. Evidence of transmural myocardial infarction is seen rarely. Exercise electrocardiograms in patients with chest pain and auscultatory evidence of floppy valves are frequently positive, exhibiting ST-segment depression despite normal coronary arteriograms. Exercise testing may precipitate arrhythmias, including multiple premature ventricular contractions and ventricular tachycardia. Thus, such tests in patients with the ballooning mitral valve syndrome should be performed with caution and careful monitoring.

Diagnostic Studies

A number of non-invasive tests may be used to establish the origin of the late systolic murmur as mitral regurgitation. Amyl nitrite administration causes the mid-late systolic extra sound to move earlier in systole, frequently merging with the first heart sound. The late systolic murmur also moves earlier in systole and may become pansystolic; its intensity does not change.

Handgrip may be very effective in evaluating these auscultatory phenomena. Handgrip may bring out a systolic extrasound which is evanescent and difficult to document.

Ultrasound examination of the mitral valve often is characteristic in the syndrome. Abnormal motion of the anterior leaflet as well as clear separation of the leaflets on the echogram are common. When present, these signs are nearly pathognomic of the ballooning posterior leaflet syndrome.

A majority of patients with either midsystolic extrasounds or late systolic murmurs are asymptomatic. Their prognosis for longevity appears independent of the extent of abnormality in the mitral leaflets. However, the occurrence of chest pain and arrhythmias is associated with considerable prognostic uncertainty. The rate of progression of the mitral regurgitation is unknown. Thus, asymptomatic patients with the auscultatory phenomena of the syndrome must be followed periodically.

When chest pain is prominent, treatment with propranolol and long-acting nitrates may be initiated. This mode of therapy has afforded significant relief in some patients. Control of arrhythmias is indicated in light of the incidence of sudden death. Ventricular extrasystoles should be treated with propranolol and digitalis, while atrial arrhythmias can often be managed effectively with digitalis alone.

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New Developments in Emergency Care

WILLIAM E. MATORY, M.D.*

The Washington Metropolitan area, with its three medical schools, its leading national legislative institutions and its large health service capabilities should be looked upon as a site that is progressive in emergency health care. Indeed, it can boast of many advances in that service. A patient can feel fairly secure in his search for quality care in true emergencies. However, the rapidity and the completeness of this care is not as universal as it should be. Non-emergency or urgent health care for the masses is still poorly delivered. The significant advances that have been achieved need to be expanded and improved. Areas with continued low standards need immediate review and change. New techniques and organizational changes must be developed to better serve the heavy, less than emergency load which faces most emergency services in the area.

The single most important need for advancement has been in the area of health personnel training. Laudable examples of this training can be seen in physician development at Howard University, physician assistants training at George Washington University and emergency medical technician training in the District of Columbia Fire Department.

At Howard University, a residency training program in Family Practice has been developed which emphasizes training in primary care. It stresses the importance of supervision and well organized training programs in the care of the ambulatory patient. This includes preventive, early detection and emergency or episodic care. It also prepares physicians to anticipate and reduce the need for episodic care, to give good care for the acutely ill and to provide in-continuity care with appropriate followup. The family physician's training prepares him for good primary care for man, woman or child in the various classical disease categories and at the various levels of urgency. It prepares the physician to man emergency facilities as well as to develop and maintain the supporting health programs which will place the emergency patient's needs in proper prospective. Supervised training in the emergency care area is also provided for all clinical students. A curriculum which includes demonstrations and practical examinations for students was first begun under auspices of the M.E.N.D. program ten years ago. This has been further developed to improve each student's expertise in emergency care and in mass episodic health care.

At George Washington University, a program is being developed which uses physician assistants to give support to physicians for the episodic care of a large number of patients. This well supervised program enables good care in depth to be provided by a team of health professionals. The physician's assistant increases a single physician's effectiveness because care for this large number of patients demands more time and effort than any physician can give by himself. This new dimension of the health team in a problem area where good supervision can be maintained promises to be an excellent solution to emergency care area staffing problems.

The Washington D. C. Fire Department Ambulance service ranks very high nationally among ambulance programs and is rapidly increasing the number of training hours for its Fire Department emergency ambulance service attendants. New attitudes in the Fire Department are developing which will insure greater "esprit de corps" among those paramedical department members who especially desire to perform this service. Assistance from the Highway Safety Act and continued support from the Department of Human Resources will further insure a top-flight emergency medical technician. Future plans include proposals to involve high quality hospital emergency rooms in the training of these emergency technicians. The city must develop a supportive health transportation system which will reduce the need for the use of emergency ambulances for non-emergency health transportation. The private ambulance technicians have generally been well trained, but should be included in the improved emergency technician program along with the fireman technician. This will more adequately insure responsible first care to the emergency patient by public and private technicians.

Emergency transportation by ambulance can be obtained by calling the City Fire Department ambulance services. Area hospitals have direct lines to the fire department and switchboard. In the Washington Metropolitan area "911" can be dialed without charge for immediate emergency ambulance services as well as for police and fire department emergencies.

Both public and private emergency transportation services are available.

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MATORY

Ten encono-van type ambulances are operated by the Washington, D.C. Fire Department. These are strategically located at various fire stations throughout the city. When dispatched they must respond to each call. They are instructed to take each patient to the nearest hospital emergency service. Patients may elect to go to another hospital of their choice, but are required to give a signed release from responsibility by the ambulance service for the time loss accordingly. Because of the quick response and free service the Fire Department Ambulance service is often misused for transportation which does not meet the usual criteria for emergencies. Private transportation is offered by a variety of private ambulance services. Three or four companies provide the major portion of private operations. Their principle activity is in the area of inter-hospital transfer which is not supported by the D.C. Fire Department Ambulance Service.

Emergency services in Category I are provided by hospitals fully equipped, prepared, and staffed in the medical and surgical specialties necessary to render resuscitative care to critically ill or injured persons of all ages. The availability of supplementary specialty services shall be prearranged with non-staff specialists and transfer of patients for specialty care should be by prior agreement with other hospitals.

Category IV emergency services must be able to give first care and then transfer the patient immediately to an emergency service area having the greatest capability.

Four area hospital emergency services meet the criteria of Category I: two university hospitals; one federal hospital and one private non-university hospital.

The area hospital emergency services vary widely in their structure, manning pattern and patient load. They also vary widely in their emergency visits to hospital bed capacity ratio: Freedmen's Hospital (Howard University)—482 Beds, 60,000 Visits; Providence Hospital—367 Beds, 50,000 Visits; George Washington University Hospital—460 Beds, 40,000 Visits; Washington Hospital Center—827 Beds, 55,000 Visits; D. C. General Hospital—1,000 Beds, 100,000 Visits.

These ratios suggest the extra work load that emergencies place on some hospitals and the need for transfer of cases which might require hospitalization. Hospital bed availability, emergency room staff capability and the multiplicity of emergencies presented are the primary indications for transfers. In many instances, transfers are still made because of financial factors.

The variety of staffing at the various emergency services ranges from hospital salaried full-time personnel in some hospitals to operation by private groups in others. The Howard University Freedmen's Hospital has the highest number of full-time licensed personnel manning its emergency care service on a 24-hour basis supported by a full complement of residents and attending staff in all major specialties. It also has developed a Family Practice Residency which has as one of its primary responsibilities the supporting of emergency services. In addition, there is an evening clinic which provides care for non-emergency evening needs.

The Washington Metropolitan Area consists of Washington, D. C. and the nearby communities of Maryland and Virginia. This area comprises approximately 2,861,123 people. Of these, 756,510 reside in Washington, D. C. proper. Approximately 800,000 emergency hospital visits are serviced each year. This service is rendered through a broad network of organizations, transportation, communication and emergency hospital programs.

The Potomac River divides Virginia and Washington, D. C. The Metropolitan Beltway, encircles Washington, D. C. and extends through Virginia and Maryland and on all sides of Washington, D. C.

A large number of automobile accident victims are serviced by hospitals located near the beltway. These include Holy Cross, Cafritz and Prince Georges County Hospitals. Those hospitals near the center of the city receive a large number of victims of social violence. This is especially true for Washington Hospital Center, Freedmen's (H.U.), Roger's Memorial and D. C. General which receive a large number of gunshot wounds.

Washington has a very dense black population in its inner city while the suburbs are inhabited mostly by the whites.

Twenty-two hospitals (plus the area military hospitals) serve the Metropolitan area. Eleven of these twenty-two hospitals are located in Washington, D. C. proper. Together these non-military hospitals service approximately 800,000 emergency patient visits annually.

The Capital City has the distinction of having the highest frequency of mass acute health problems because of its frequency as a site for mass demonstrations. The D. C. Health Mobilization Unit has been able to develop and maintain the needed coordination and cooperation of the area health institutions and personnel to insure adequate coverage and response to this need.

The Mayor's Advisory Committee on Emergency Medical Services includes representatives from the D. C. Medical Society, Medico-Chirurgical Society, Hospital Council of the National Capital Area, Health Facilities Planning Council of Metropolitan Washington, Department of Human Resources, Fire Department Ambulance Service, D. C. Police Department, Highway Safety Council, and the local branch of the American College of Surgeons. This committee is responsible for advising the community on aspects of emergency health care.

The Highway Safety Act provides several standards for federal support of programs related to safety on federal highways. One of the standards, "Standard 11" provides funds for each major city to support a coordinator of training for emergency care personnel and equipment. It also provides funds to facilitate the coordination of emergency health care. The District of Columbia program has advanced to the organization required and has begun to implement training, data collection and equipment. It has also developed an ordinance which establishes the level of training for ambulance attendants (emergency medical technicians).

The American College of Emergency Physicians is the national organization for physicians involved in emergency care. This organization has done much to emphasize the importance of this service and to improve the Physician Staffing of emergency medical services throughout the country. The University Association of Emergency Medical Services has as one of its primary responsibilities, the improvement of physician training in emergency care. This organization is developing standards for medical students and physician development.

The previous discussion reveals many advances. However, the complete picture of health care in our emergency facilities is not so bright. The large number of patients with non-urgent problems who present to Emergency Care Area must be reduced, managed better and given better follow-up care. The development of more innovative programs and the adoption of these programs by more emergency facility areas are imperative.

REMARKS BY LOUIS GILLESPIE, M.D.†

Dr. Matory has presented some thoughts about Emergency Care in the District of Columbia. He points to a number of programs being developed to supply trained manpower to the existing emergency services locally. Unfortunately, these training programs are only in the developmental stage presently, and some time will yet pass before their fruits are harvestable.

Dr. Matory's remarks also touch upon what I believe strongly is a lack of coordination in the development of these new programs as well as the optimal care of those patients presenting to the emergency facilities in the District. We have Mayor's Task Forces with multiple representation; the Department of Human Resources has ideas and recommendations; the private hospitals and the university hospitals have problems, committments, and plans; and the D.C. Fire Department has programs in planning as well as morale and budgetary problems. The currently operational subcommittees of the D.C. Hospital Conference of 1972 will shortly be adding to the planning input. I ask from where will come the coordination of all of these groups and from where will come the leadership finally to implement the best of all the planning input? It is just such fragmentation of plans, programs, and services which has led to the present stage of inefficient and overly costly health care delivery–acute and chronic–in Washington.

As Dr. Matory indicates, the care of the acute medical emergency is quite satisfactory in those Category I hospital emergency services. But, as he points out, the two glaring areas which require urgent upgrading are the ambulance transport services and the services rendered to those patients who come to the emergency services with less than acute medical problems.

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The Clinical Utility of In-Vitro Tests of Thyroid Function

JAY R. SHAPIRO, M.D.* AND RICHARD C. REBA, M.D.†

Unfortunately, dysfunction of the thyroid is like malfunction of many organs in that one cannot make a valid diagnosis of "too much" or "too little" without resorting to several laboratory tests. The proliferation of thyroid function tests with similar sounding names, such as "T-4 by column", or "T-4 by Murphy-Pattee" has caused confusion for the clinician. The "T-3 test" [resin uptake] so commonly used today has also been confused with those newer procedures which determine the concentration of triiodothyronine [T-3] in plasma. What then is the difference between the resin T-3 uptake and the determination of T-3 content in blood?

The resin T-3 uptake is an *in-vitro* test which indirectly assesses thyroid function by the degree to which serum thyroid binding proteins are saturated with circulating hormone. 99.96% of T-4 is reversibly bound to three plasma proteins: thyroxine binding globulin [TBG], thyroxine binding pre-albumin [TBPA], and albumin, and therefore, 0.04% remains free. The free fraction is the metabolically active component and is directly proportional to thyroxine [T-4] concentration and indirectly proportional to the number of unsaturated thyroid binding sites [UTBG]. TBG, which binds both T-4 and T-3, has a low total capacity, but high specificity, and binds the former much more avidly than the latter. On TBG, both T-4 and T-3 bind to identical sites, but as stated, T-3 is much less avidly bound and this can be used to assess the degree to which hormone binding sites are occupied circulating thyroxine. Serum is enriched with a small quantity of I-125 labeled T-3, which saturates unoccupied binding sites of the patient's TBG. The serum is then exposed to insoluble particulate material (resin or charcoal) which separates protein bound radioactive hormone from any of the added label not attached to binding sites. The added labeled T-3 apportions itself between the binding sites and the resin and so serves as a marker to indicate the number of TBG binding sites unoccupied by circulating hormone, i.e., the amount of hormone bound to the resin is indirectly proportional to the number of TBG sites occupied by hormone. Triiodothyronine is used in preference to T-4 because being bound less firmly, it gives higher resin values than would T-4 which serves to reduce the counting error. The less label bound to the patient's proteins, the higher the thyroid hormone production rate and visa versa. As will be discussed, the concentration of hormone and TBG in the patient's plasma will influence the results of these tests.

The cause of several common misconceptions should now be apparent. T-3 resin uptake clearly does not measure thyroid uptake, does not measure T-3 concentration, and is not related to T-3 metabolism. Rather, as noted above, this *in-vitro* determination is an indirect measurement of the unsaturated binding sites of TBG, and in a way, of the amount of TBG present.

One might ask, since the free thyroxine concentration [FT-4] is the metabolically active hormone, why not just measure this parameter? Presently FT-4 is impossible to measure directly and, the indirect estimations are difficult, tedious, and time-consuming and not yet appropriate for the clinical laboratory. Since FT-4 is proportional to T-4/UTBG and the T-3 resin uptake is related to UTBG, it can be demonstrated mathematically that the product of T-3 uptake and T-4 is related to FT-4. Thus, this product should result in a good approximation of FT-4 and, therefore, true thyroid activity. This value, variously termed T-7, T-12, FT-Index, is particularly useful when T-3 resin uptake and T-4 measurements are divergent, as from non-thyroidal illness or from certain drugs. Fortunately, in thyroid disease, these tests are usually not divergent, but are both elevated in hyperthyroidism and decreased in hypothyroidism. [Table 1]

However, for reasons unrelated to thyroid function, FT-4 is frequently increased in seriously ill patients or after severe trauma [Table 2]. This is particularly unfortunate, since frequently it is in such patients that one is dependent on laboratory help for diagnosis. Therefore, the T-7, T-12, or

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FT-Index is best used for the assessment of thyroid function during pregnancy, in patients receiving sex hormones, (e.g. birth control pill), or drugs which interfere with T-4 binding of TBG (e.g. dilantin), and in patients suspected of having abnormalities of their thyroxine binding proteins [See Table 3].

It is now possible to measure the total concentration of T-3 in plasma, and to estimate the minute quantity which is not protein bound, but unbound ("free"), and therefore, that which is metabolically active. However, the determination of T-3 in plasma by either gas or liquid chromatography, immunoassay, or protein binding techniques has not only brought new data to light about hormone secretion, but has documented the presence of patients with hyperthyroidism due to oversecretion of T-3 rather than T-4, ie. "T-3 toxicosis", in the presence of a normal or low T-4 level. The determination of total T-3 in plasma is available in only a few laboratories today, but with refinement of techniques it should be widely available within a year or two.

TABLE 1

	Clinical State	[T-4]	T-3 Uptake	TBG	FT-4
Normal pregnancy (or Mole), birth control pill, estrogens feminizing tumors	euthyroid	↑	↓	↑	N
Threatened, spontaneous abortion	euthyroid	N	N	N	N
Pregnancy with thyrotoxicosis	hyperthyroid	↑	N	↑	↑
Pregnancy with hypothyroidism	hypothyroid	↓N	↓	↑	↓
Androgens, anabolic steroids, masculinizing disorder	euthyroid	↓	↑	↓	N
Hypoproteinemia, e.g., severe nephrosis, debilitating diseases	euthyroid	↓	↑	↓	N
Drugs that compete with T-4 for TBG sites, e.g., dilantin	euthyroid	↓	↑	N	N
Thyrotoxicosis	hyperthyroid	↑	↑	N	↑
Myxedema, untreated	hypothyroid	↓	↓	N	↓
With T-4 therapy	euthyroid	↑N	↓N	N	↑N
With T-3 therapy	euthyroid	↓	↓N	N	↓N

↑ = Increased.
N = Normal.
↓ = Decreased.

TABLE 2: Some Common Conditions that May Result in an Elevated FT-4

1. Malignancy, e.g., lung, stomach; multiple myeloma, lymphoma, leukemia
2. Inflammation, e.g., ulcerative colitis, tuberculosis, rheumatic heart disease, SBE, collagen vascular disease, infectious mononucleosis
3. Others, e.g., heparin administration, Wilson's Disease, amyloid, complicated diabetes mellitus

TABLE 3: Conditions that do not Affect T-3 Uptake Test

1. Eumetabolic thyroid disease, e.g., multinodular/multicystic goiter, thyroid cancer
2. Other endrocrinopathies, e.g., Addison's Disease, Cushing's Disease, pituitary disorders
3. Exogenous iodides, including x-ray contrast media
4. Hypertension, congestive heart failure, anxiety
5. Others: mercurial drugs, most instances of liver diseases and anemia

Reference

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Abbreviations
T-3 = Triiodothyronine
T-4 = Thyroxine
FT-4 = "free" or unbound thyroxine
TBG = Thyroid binding globulin
UTBG = Unbound thyroid binding globulin
FT-Index = "Free Thyroxine" Index

The Good Samaritan Laws

Over one-third of those physicians replying in recent surveys unequivocally stated they would not render emergency care to persons involved in an accident, even though Section 5 of the Principles of Medical Ethics of the American Medical Association provides that in an emergency a doctor should render service to the best of his ability. Their reasoning was based upon their fear of becoming involved in malpractice litigation which could be damaging to their reputation.

In an attempt to allay this fear and encourage doctors and nurses to give aid in such situations, over 30 states (including Maryland and Virginia) and the District of Columbia have adopted Good Samaritan Statutes. It is interesting to note that through August 15, 1969, no medical malpractice case construing such Good Samaritan Statutes involving emergency aid rendered by a physician had reached a state appellate court.

Assume that while driving home late one evening, you notice a man covered with blood lying face down in the middle of the road, his automobile crunched into a telephone pole. Morally, you want to stop and help, but legally must you? If you do stop, what potential liabilities will attach, and what protection will be afforded you—the good samaritan doctor?

Common Law

The general common law rule is that there is no legal duty to be a good samaritan and aid one who is injured, except where a special relationship exists, such as the person causing the accident being responsible for the condition of the injured stranger. However, once a doctor goes to the assistance of an injured person, he is potentially liable. Generally, so long as the good samaritan does not place the injured person in a worse condition than the condition prior to his rendering assistance, no liability will attach. Of course, this is a vague standard of care which is difficult to define.

Applying the above to our factual setting, a doctor is not legally bound to aid the injured man; however, upon initiating any type of first aid, he must not jeopardize the victim's situation.

District of Columbia

The District of Columbia Good Samaritan Statute (§2-142, D. C. Code) provides that any physician or registered nurse shall not be liable for any act or omission while rendering medical care at the scene of an accident or other medical emergency, provided that: 1) he acts in good faith, 2) he is not grossly negligent, 3) he performs such services without expectation of receiving or intending to seek compensation, and 4) the emergency occurs in the District of Columbia and outside a hospital.

The statute applies to *any* physician or registered nurse, not only those registered in the District of Columbia; its protection is also extended to osteopaths. The statute applies whether the physician is called to the scene of the emergency or just happens upon it.

The statutory standard of care is two-fold: presence of good faith and absence of gross negligence. Neither of these standards is defined by the statute. "Good faith" is generally that state of mind denoting honesty of purpose and professional integrity and a reasonable opinion that the situation calls for immediate care. It would appear that whenever a doctor renders emergency care, he is doing so in good faith; this is perhaps the reason why this standard is parenthetically expressed in the statute. "Gross negligence," on the other hand, may be said to be the failure to use care in such a manner as to be completely indifferent to the consequences of the act. These standards of care are substantially equal to the common law requirement not to place the injured person in a worse condition than that in which he was found.

Another provision of the statute is that the services must be performed gratuitously. Assuming that you do stop to assist the injured man, does this mean that you are not entitled to compensation for the supplies used? Under common law rules, nearly every jurisdiction provides for the recovery of these expenses. But the attempt to recover these costs could cause the physician to lose the protection of the statute. Therefore, it would appear to be advisable in such circumstances that the physician refrain from attempting to collect compensation for emergency medical care. In this way, the gratuity test will not be violated.

The statute speaks of medical care and assistance at the scene of an accident or other medical emergency. Generally, an emergency is a sudden unexpected occurrence which demands immediate action. Our factual situation would fall within this statutory requirement.

But what of the duration of the emergency? The statute refers only to "medical care and assistance at the scene of an accident or other medical emergency." Will you be protected if you take the injured man to the hospital? The statute is silent on this point so that one cannot state the answer with certainty. There appears to be no reason why this act should not be protected. Other states (such as Virginia) with similar statutes specifically include the transporting of the injured person to the hospital as part of the emergency. One important point: once the injured person is removed from the scene of the accident, he should be transported *only* to a hospital or other medical facility and not to the home of a friend or a relative. In 1969, the New Mexico Supreme Court, construing a Good Samaritan Statute similar to the D. C. Statute, held that a person (not a physician) who was transporting an accident victim to a motel was not providing emergency medical care within the meaning of the statute.

Maryland

The Maryland statute is as ambiguous as that of the District because none of the requirements is defined. Article 43, §132 of the Annotated Code of Maryland (1957) provides that any physician, registered nurse or licensed practical nurse shall not be liable for any professional act or omission while rendering medical care, aid or assistance in Maryland under emergency conditions at or near the scene of an accident or other occurrence, provided that: 1) he is not grossly negligent, 2) he charges no fee or compensation, and 3) the emergency is not in a hospital.

The same principles discussed under the District of Columbia statute should apply in Maryland regarding recovery of expenses, and the locus and duration of the emergency. However, there are some differences in the Maryland statute. First, the Maryland statute includes licensed practical nurses (and, in certain situations, members of fire departments or volunteer ambulance and rescue squads). Second, there is no mention of good faith as a standard of care. As noted above, this absence probably connotes that a physician rendering emergency aid is doing so in good faith.

Virginia

The law in Virginia has a somewhat wider scope than those of the District and Maryland. §54-276.9 of the Code of Virginia (1950) provides that *any person* who renders emergency care to an injured person at the scene of an accident or fire, or enroute therefrom to any hospital, medical clinic or doctor's office, shall not be liable for acts or omissions resulting therefrom, provided that: 1) he acts in good faith, and 2) he performs the act or acts without compensation.

Interestingly enough, another portion of the statute denies immunity from liability arising out of the operation of a motor vehicle.

Prior to 1964, the statute applied only to physicians. In that year, it was amended to include dentists. In 1971, it was revised to protect *all* persons, including Virginia residents and non-residents alike.

The only standard of care required is the presence of good faith, there being no mention of abstention from gross negligence.

The major difference in the Virginia statute is the duration of the emergency. It includes not only the scene of the emergency, but also the transporting of the injured person to a place where adequate medical treatment is available. Thus, if in our hypothetical situation you render aid to the injured man, the statutory protection will be available not only at the scene of the accident, but also while transporting him to a hospital, medical clinic or doctor's office.

Finally, one section of the statute denies immunity from liability arising from the operation of a motor vehicle. Whether this section denies immunity to the good samaritan who renders emergency care to a person injured as a result of that operation of a motor vehicle by the good samaritan which caused the emergency to arise in the first place or denies immunity to the good samaritan who is involved in an automobile accident while transporting the injured person from the scene of the original accident to a proper medical facility is somewhat unclear. However, if it

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the 3 phases of Eve

All women are not equal in their endogenous hormonal output. And, while all oral contraceptives are fundamentally effective, they exhibit **differences in their activity levels and estrogen-progestogen ratios that affect different women differently**—in both short and long-term use. Some brands may be insufficient for the woman's needs or else may exceed them.

Searle offers a family of O.C. products that covers the range of women's needs to help you provide the right pill for the right woman at the right time.

the Ovulen phase

Most women* with a **balanced hormone profile and normal menses** do best on a middle-of-the-road pill that is neither estrogen dominant nor strongly progestogen dominant.

(*Typical clues—normal body build and breasts, feminine appearance, healthy skin and hair. Vaginal cytology slide—balanced "pink and blue.")

Some women having problems on other O.C.s might do well on Ovulen.

Ovulen has a distinctive hormonal balance that combines moderate estrogenic activity with a slight progestogen dominance. It has an excellent record of patient acceptance.

Ovulen®

Each white tablet contains: ethynodiol diacetate 1 mg/mestranol 0.1 mg.

References. **1.** Editorial Oral Contraceptives Which Pill for Which Patient? Patient Care *3*:90-115 (Feb.) 1969 and *4*:135-145 (June 15) 1970 **2.** Greenblatt, R. B Progestational Agents in Clinical Practice, Med Sci *18* 37-49 (May) 1967 **3.** Kistner, R W Gynecology: Principles and Practice, ed 2, Chicago, Year Book Medical Publishers, 1971 **4.** Kistner, R. W: The Pill: Facts and Fallacies About Today's Oral Contraceptives, New York, Delacorte Press, 1968. **5.** Nelson, J H: Clinical Evaluation of Side Effects of Current Oral Contraceptives, J Reprod Med. 6:50-55 (Feb.) 1971 **6.** Orr, G W Oral Progestational Agents. Therapy and Complications, S Dakota J Med. *22*:11-17 (Jan.) 1969

SEARLE

For brief summary of prescribing information, see following page.

the Demulen phase

Many women* who secrete **more estrogen** than most do well on a pill with lower estrogen activity and an increased progestogen overbalance.

(*Typical clues—shorter, plumper, full-breasted, with glowing skin and no wrinkles. Vaginal cytology slide "pink.")

Some women with special conditions that may be aggravated by higher estrogen-activity products may do better on this ratio.

Demulen combines minimal estrogenic activity with a moderate ratio of progestogen overbalance. It is particularly well suited to the young when low-dose (activity) is preferred. Demulen offers little risk of the most potent progestogen side effects; early breakthrough bleeding is often transient.

Demulen®

Each white tablet contains ethynodiol diacetate 1 mg/ethinyl estradiol 50 mcg.
Each pink tablet in Ovulen-28® and Demulen® 28 is a placebo containing no active ingredients.
Both Ovulen and Demulen are available in 21- and 28-pill schedules.

the Enovid-E phase

Some women* who secrete **less estrogen** than most do best on a pill with a moderate estrogen overbalance.

(*Typical clues—oily complexion, acne, hirsutism, masculinity, flat chest. Vaginal cytology slide—"blue.")

Patients with estrogen deficiency may show:

premenopausal syndrome
early-cycle bleeding
scanty menses
intermittent depression
increased appetite
steady weight gain
vaginal candidiasis

Enovid-E not only provides increased estrogenic activity with low progestogen activity, but also contains the only progestogen that is not antiestrogenic. Therefore it offers less risk of high-dose progestogen side effects.

Enovid-E®

Each tablet contains norethynodrel 2.5 mg/mestranol 0.1 mg.

Oral contraceptives are complex medications and, after reference to the prescribing information, should be prescribed with discriminating care.

for the 3 phases of Eve:

a family of O.C. products

Ovulen®

Each white tablet contains:
ethynodiol diacetate 1 mg./mestranol 0.1 mg.

Demulen®

Each white tablet contains:
ethynodiol diacetate 1 mg./ethinyl estradiol 50 mcg.

Each pink tablet in Ovulen-28® and Demulen®-28 is a placebo, containing no active ingredients.

Actions—Ovulen and Demulen act to prevent ovulation by inhibiting the output of gonadotropins from the pituitary gland. Ovulen and Demulen depress the output of both the follicle-stimulating hormone (FSH) and the luteinizing hormone (LH).

Special note—Oral contraceptives have been marketed in the United States since 1960. Reported pregnancy rates vary from product to product. The effectiveness of the sequential products appears to be somewhat lower than that of the combination products. Both types provide almost completely effective contraception.

An increased risk of thromboembolic disease associated with the use of hormonal contraceptives has now been shown in studies conducted in both Great Britain and the United States. Other risks, such as those of elevated blood pressure, liver disease and reduced tolerance to carbohydrates, have not been quantitated with precision.

Long-term administration of both natural and synthetic estrogens in subprimate animal species in multiples of the human dose increases the frequency of some animal carcinomas. These data cannot be transposed directly to man. The possible carcinogenicity due to the estrogens can be neither affirmed nor refuted at this time. Close clinical surveillance of all women taking oral contraceptives must be continued.

Indication—Ovulen and Demulen are indicated for oral contraception.

Contraindications—Patients with thrombophlebitis, thromboembolic disorders, cerebral apoplexy or a past history of these conditions, markedly impaired liver function, known or suspected carcinoma of the breast, known or suspected estrogen-dependent neoplasia and undiagnosed abnormal genital bleeding.

Warnings—The physician should be alert to the earliest manifestations of thrombotic disorders (thrombophlebitis, cerebrovascular disorders, pulmonary embolism and retinal thrombosis). Should any of these occur or be suspected the drug should be discontinued immediately.

Retrospective studies of morbidity and mortality conducted in Great Britain and studies of morbidity in the United States have shown a statistically significant association between thrombophlebitis, pulmonary embolism, and cerebral thrombosis and embolism and the use of oral contraceptives. There have been three principal studies in Britain[1-3] leading to this conclusion, and one[4] in this country. The estimate of the relative risk of thromboembolism in the study by Vessey and Doll[3] was about sevenfold, while Sartwell and associates[4] in the United States found a relative risk of 4.4, meaning that the users are several times as likely to undergo thromboembolic disease without evident cause as nonusers. The American study also indicated that the risk did not persist after discontinuation of administration and that it was not enhanced by long-continued administration. The American study was not designed to evaluate a difference between products. However, the study suggested that there might be an increased risk of thromboembolic disease in users of sequential products. This risk cannot be quantitated, and further studies to confirm this finding are desirable.

Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions medication should be withdrawn.

Since the safety of Ovulen and Demulen in pregnancy has not been demonstrated, it is recommended that for any patient who has missed two consecutive periods pregnancy should be ruled out before continuing the contraceptive regimen. If the patient has not adhered to the prescribed schedule the possibility of pregnancy should be considered at the time of the first missed period.

A small fraction of the hormonal agents in oral contraceptives has been identified in the milk of mothers receiving these drugs. The long-range effect to the nursing infant cannot be determined at this time.

Precautions—The pretreatment and periodic physical examinations should include special reference to the breasts and pelvic organs, including a Papanicolaou smear since estrogens have been known to produce tumors, some of them malignant, in five species of subprimate animals. Endocrine and possibly liver function tests may be affected by treatment with Ovulen or Demulen. Therefore, if such tests are abnormal in a patient taking Ovulen or Demulen, it is recommended that they be repeated after the drug has been withdrawn for two months. Under the influence of progestogen-estrogen preparations preexisting uterine fibromyomas may increase in size. Because these agents may cause some degree of fluid retention, conditions which might be influenced by this factor, such as epilepsy, migraine, asthma, cardiac or renal dysfunction, require careful observation. In breakthrough bleeding, and in all cases of irregular bleeding per vaginam, nonfunctional causes should be borne in mind. In undiagnosed bleeding per vaginam adequate diagnostic measures are indicated. Patients with a history of psychic depression should be carefully observed and the drug discontinued if the depression recurs to a serious degree. Any possible influence of prolonged Ovulen or Demulen therapy on pituitary, ovarian, adrenal, hepatic or uterine function awaits further study. A decrease in glucose tolerance has been observed in a significant percentage of patients on oral contraceptives. The mechanism of this decrease is obscure. For this reason, diabetic patients should be carefully observed while receiving Ovulen or Demulen therapy. The age of the patient constitutes no absolute limiting factor, although treatment with Ovulen or Demulen may mask the onset of the climacteric. The pathologist should be advised of Ovulen or Demulen therapy when relevant specimens are submitted. Susceptible women may experience an increase in blood pressure following administration of contraceptive steroids.

Adverse reactions observed in patients receiving oral contraceptives—A statistically significant association has been demonstrated between use of oral contraceptives and the following serious adverse reactions: thrombophlebitis, pulmonary embolism and cerebral thrombosis.

Although available evidence is suggestive of an association, such a relationship has been neither confirmed nor refuted for the following serious adverse reactions: neuro-ocular lesions, e.g., retinal thrombosis and optic neuritis.

The following adverse reactions are known to occur in patients receiving oral contraceptives: nausea, vomiting, gastrointestinal symptoms (such as abdominal cramps and bloating), breakthrough bleeding, spotting, change in menstrual flow, amenorrhea during and after treatment, edema, chloasma or melasma, breast changes (tenderness, enlargement and secretion), change in weight (increase or decrease), changes in cervical erosion and cervical secretions, suppression of lactation when given immediately post partum, cholestatic jaundice, migraine, rash (allergic), rise in blood pressure in susceptible individuals and mental depression.

Although the following adverse reactions have been reported in users of oral contraceptives, an association has been neither confirmed nor refuted: anovulation post treatment, premenstrual-like syndrome, changes in libido, changes in appetite, cystitis-like syndrome, headache, nervousness, dizziness, fatigue, backache, hirsutism, loss of scalp hair, erythema multiforme, erythema nodosum, hemorrhagic eruption and itching.

The following laboratory results may be altered by the use of oral contraceptives: hepatic function: increased sulfobromophthalein retention and other tests; coagulation tests: increase in prothrombin, Factors VII, VIII, IX and X; thyroid function: increase in PBI and butanol extractable protein bound iodine, and decrease in T^3 uptake values; metyrapone test and pregnanediol determination.

References: 1. Royal College of General Practitioners: Oral Contraception and Thrombo-Embolic Disease, J. Coll. Gen. Pract. *13:*267-279 (May) 1967. **2.** Inman, W. H. W., and Vessey, M. P.: Investigation of Deaths from Pulmonary, Coronary, and Cerebral Thrombosis and Embolism in Women of Child-Bearing Age, Brit. Med. J. *2:*193-199 (April 27) 1968. **3.** Vessey, M. P., and Doll, R.: Investigation of Relation Between Use of Oral Contraceptives and Thromboembolic Disease. A Further Report, Brit. Med. J. *2:*651-657 (June 14) 1969. **4.** Sartwell, P. E.; Masi, A. T.; Arthes, F. G.; Greene, G. R., and Smith, H. E.: Thromboembolism and Oral Contraceptives: An Epidemiologic Case-Control Study, Amer. J. Epidem. *90:*365-380 (Nov.) 1969.

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Actions—Enovid-E acts to prevent ovulation by inhibiting the output of gonadotropins from the pituitary gland. Enovid-E depresses the output of both the follicle-stimulating hormone (FSH) and the luteinizing hormone (LH).

Indication—Enovid-E is indicated for oral contraception.

The *Special Note, Contraindications, Warnings, Precautions* and *Adverse Reactions* listed above for Ovulen and Demulen are applicable to Enovid-E and should be observed when prescribing Enovid-E.

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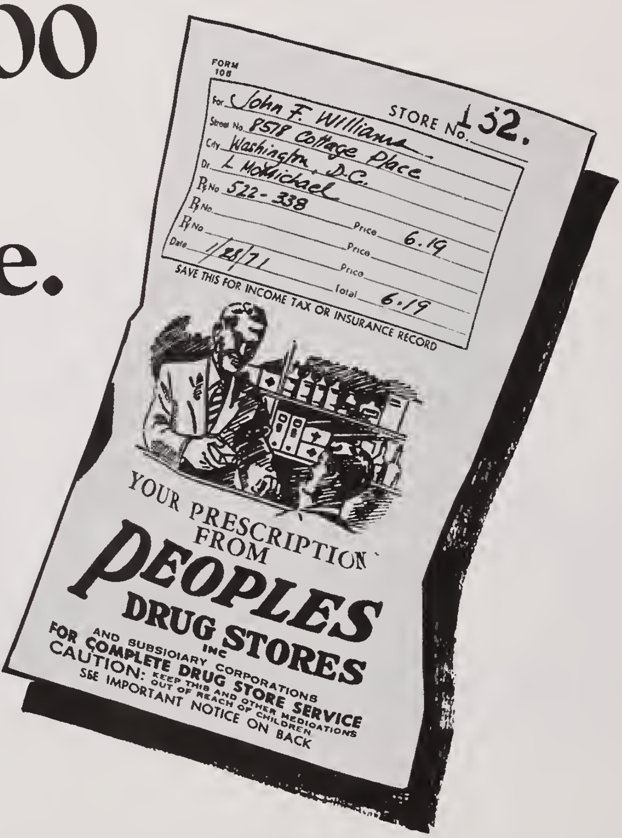
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pertains to the transporting of the injured person to a medical facility, it would contradict the first part of the statute which specifically protects the act of transportation. Thus, it would seem that the denial of immunity from liability arising out of the operation of a motor vehicle should only apply to that operation of the motor vehicle which caused the emergency to arise in the first place.

Conclusion

The scope of this article has been limited to the protection afforded a good samaritan doctor who renders emergency aid. Compliance with the statutory mandate should provide a defense to the physician involved in malpractice litigation resulting from his good samaritan act. However, it should be noted that the statute does not prevent the institution of such suit by the injured party.

ROBERT A. ROMBRO, J.D.

Edited by Stefan F. Tucker of Arent, Fox, Kintner, Plotkin & Kahn, Washington, D.C.

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d.c. MENTAL HEALTH ADMINISTRATION

Forest Haven—Current Programs and Treatment

"The public demands programs and treatment for these children," said the television narrator. "These children need the professional help of psychiatrists and psychologists," stated the author of an article in a national magazine recently. They were talking about Forest Haven, the District Institution for the retarded.

Of course there are programs at Forest Haven for many of the residents. There is a public school serving over two hundred of the school age residents. It is adequately manned by highly trained teachers, over half of whom have master's degrees. The school's physical plant is clean, modern and efficient in design.

Thanks to other programs at Forest Haven, 73 residents were discharged to the community last year. Another 100 earn their way by working on the grounds at Forest Haven, while others work in the local Marriott Inns, in the stock room and kitchen at Lord and Taylor, at the Beltsville Farms, and so forth. They are not languishing at Forest Haven, "waiting to die" as another writer put it last year. In fact, 46 residents live in and maintain their own apartments on the grounds of Forest Haven in buildings where staff members used to live.

Another program involves training lower functioning residents to accomplish for themselves those tasks that were formerly performed by the harried staff or the parents of the residents during weekend visits at home. Such tasks as dressing themselves, taking care of their own toilet needs, feeding themselves, and basic interpersonal skills.

In this latter program, the operant conditioning procedures of the Behavioral Psychologist are used, but with only positive reinforcers brought into play; M&M candy and tokens for the brighter trainees, and candy alone for the others. Among other things, they can purchase toys with their tokens, and save their candy for a private party later or share it with friends. Near-miraculous results are obtained with such training, though the end tasks are still short of those often desired by the public. The complexity of the tasks learned has greater range than the scientific community would have thought possible even five years ago, but it will never reach the aspirations of a parent. Just like with normal children.

All residents at all functioning levels could take advantage of behavioral training. One catch is that such training involves immediate reinforcement each time the desired behavior is emitted. Another, more desirable situation from the standpoint of training efficiency, demands that reinforcers later be given on a random schedule, or on some other set schedule of one kind or another. The problem is that no matter which training paradigm is used, someone must see the behavior—actually count it—each time it occurs in order to manage the reinforcement schedule effectively. And learning will not occur in the absence of reinforcement. Worse still, neuroses can be learned if the contingencies of reinforcement are inconsistent. Just like in normal children in normal homes.

The reader can see the ultimate problem. The teacher-student ratio must be very small. Indeed, the suggested ratio is 1:1, and even in groups where the ratio is one teacher to 15 students, as is currently the case at Forest Haven in these training groups, many students must be relatively ignored while a few are trained at any given time.

At Forest Haven one psychologist was working with three groups of 46 trainable residents as late as December of last year. He is no longer at Forest Haven. Few psychologists want to work with this difficult group of students, and there is no foreseeable squeeze on psychologists in the job market in the nation, so even if a hundred or so slots were made available for psychologists at Forest Haven it is likely that they would remain vacant. 100 psychologists or other teachers trained in behavior modification principles would be a nominal start in achieving effectiveness, by the way.

One now comes full circle back to a basic philosophical question. Will the public, even a stirred up public, provide the funding support for the real programs that they and the journalists demand? The answer is, not likely. Not while returning veterans demand job training. Not while welfare mothers and their children are hungry and in need of housing and clothing. Not when the public schools are in disarray, and the crime rate at an unconscionable high.

Earlier in this series the comment was made that politicians use funds to oil squeaky wheels. They are not necessarily motivated by humanistic philosophies as such, nor by a concentrated, consistent view of what is best for society. But a socio-political philosophy would encourage them to spend public funds on Forest Haven residents only after the needs of the more socially productive elements were met. This is the priority Congress has maintained until recently. Assume that Congress became less stressed by the crime in our streets, the overcrowded courts, the hungry people in the ghettoes of our city, rising protests over ever increasing taxes, etc., and delegated large amounts of money to programs at Forest Haven. Significant problems would remain.

For one thing, 200 of our residents are totally helpless, bedridden patients of all ages with I.Q.'s so low they are unmeasurable. Another 200 are ambulatory patients but at a similarly low functioning level. All must have diapers, and someone to change them regularly, even though the resident is 50 years of age or older. The demand is for programs and recreation for these patients. Research has not indicated what effective programs for these patients would be. In addition, research has shown us little or nothing concerning effective recreation for these residents. Yet this is another public demand, according to the journalists and a number of parents.

We are moving at Forest Haven, but into the unknown. For example, 200 residents were moved from an overcrowded, overheated, ill lighted and improperly ventilated building late last year and into a new, well lighted, well ventilated, uncrowded building with a cheery atmosphere and smaller, more intimate groupings of patients into modular living units. The colors are bright, with yellows, oranges and reds predominating. The dining areas are small, comfortable and uncrowded. Each living unit has an enclosed courtyard where the residents can gather or play in warm weather, getting fresh air and sunlight not available in their former quarters. Sounds ideal.

As it turns out, the prevailing colors are what the architect and his staff thought would appeal to the mentally retarded. We have no information on color preferences among this group, and the colors chosen may ultimately be deleterious to their emotional wellbeing. It is an unresearched subject.

The residents are given beds to sleep on that are the height of beds in private homes. The floor beneath them is hard, and some tumble out of their beds from time to time. What would be the effect of placing their mattresses at floor level to lessen or delete the possibility of them injuring themselves when they roll off the edge? Another unknown. Many feel that it would be a step backward from readying them to resume a position in normal homes with the usual beds. Certainly the visiting public would be offended, and journalists would point out that our residents were "sleeping on the floor." So the beds remain at the "proper height" and some of the more restless residents, especially those with a history of falling to the floor, are provided with restraints to avoid further injuries. Now the cry has gone up that patients are tied into bed. This is true. Some of them are restrained safely in "normal beds." Research has not indicated any ultimate deleterious effects on the residents of sleeping closer to the floor or of being restrained in beds of normal height. Certainly diapers of residents weighing 150 pounds or more are more easily changed on the 200 who are permanently in beds that are higher off the floor.

As for recreation, typical recreation involving motor coordination and/or prolonged attention are out. What is available to them is another unresearched question. What is their interest level in things external to themselves? What is their potential for boredom when sitting quietly? While we await some of these answers, the residents at least have a lawn to play on now. And what is the reaction of the public?

"A concrete jail," noted one journalist, referring to the enclosed courtyards that many of us would have paid thousands of dollars to have constructed around our own yards for privacy. We do not need the private courtyards at Forest Haven, of course. If we can hire responsible adults to accompany them, we can let them play on the grounds. But we will need a lot more adults, and the somewhat artificial effect of having an adult trailing at all times during recreation activities has not been researched. Certainly it would not prepare them to live in the city.

There are many questions, then, concerning possible programs and training for the mentally deficient. We could not wait for scientists to answer all of our questions before we moved from the type of care provided these residents in our recent past. Unfortunately, researchers are few in number and they have their own priorities. Researching many of the subjects listed here is not high on their list of things to do.

So we will move on, and in so doing will make mistakes. And we will do it without proper funding, or staff, or equipment, or plant. But we will continue to move, bending every resource to the task of helping the residents achieve the happiest, most satisfying, most productive life possible. And we will be criticized every step of the way, for that is the nature of public service. But we wouldn't have it otherwise in a democracy.

In the next issue the programs we would like to pursue at Forest Haven will be outlined.

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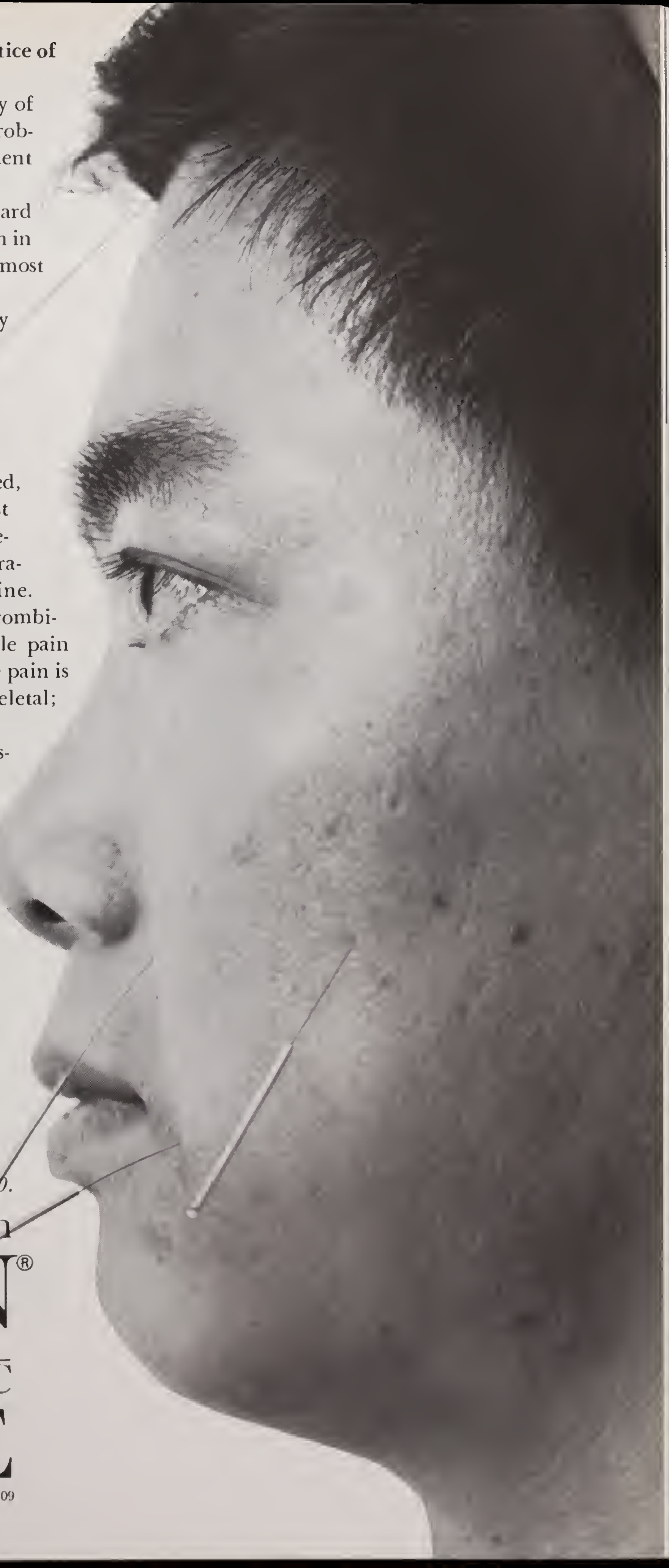

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Warnings: Use during pregnancy is to be avoided.

Precautions: 1. *Starvation Ketosis:* This must be differentiated from "insulin lack" ketosis and is characterized by ketonuria which, in spite of relatively normal blood and urine sugar, may result from excessive phenformin therapy, excessive insulin reduction, or insufficient carbohydrate intake. Adjust insulin dosage, lower phenformin dosage, or supply carbohydrates to alleviate this state. **Do not give insulin without first checking blood and urine sugar.**
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ıould also be made of electrolytes, pH, and the
ıctate-pyruvate ratio. The drug should be with-
rawn and insulin, when required, and other
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the appearance of any metabolic acidosis.

3. *Hypoglycemia:* Although hypoglycemic reactions are rare when phenformin is used alone, every precaution should be observed during the dosage adjustment period particularly when insulin or a sulfonylurea has been given in combination with phenformin.

Adverse Reactions: Principally gastrointestinal; unpleasant metallic taste, continuing to anorexia, nausea and, less frequently, vomiting and diarrhea. Reduce dosage at first sign of these symptoms. In case of vomiting, the drug should be immediately withdrawn. Although rare, urticaria has been reported, as have gastrointestinal symptoms such as anorexia, nausea and vomiting following excessive alcohol intake.
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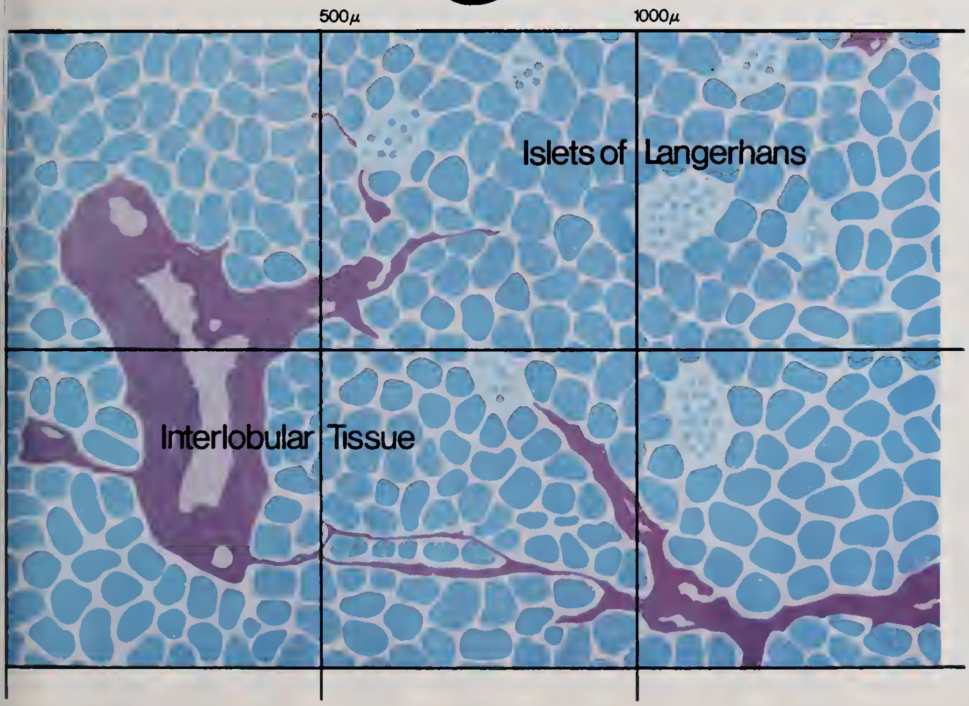

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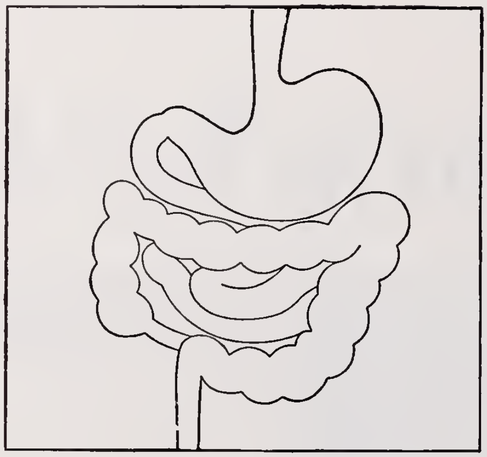

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d.c. HEALTH NOTES

Last Class For Capital City School of Nursing

Capital City School of Nursing closed its doors after almost 100 years. It was organized in 1877 as the first school of nursing in the District of Columbia and was known as the Washington Training School for Nurses.

During the years, almost 1,600 students graduated and all have passed the State Board examination for licensure.

The School provided nursing staff for D. C. General Hospital. Of the 29 students who graduate this month, 24 have accepted employment with D. C. General Hospital.

The number of diploma schools in the United States has decreased from some 1,200 to less than half that amount. This is due to the change in educational requirements for nurses during the last decade. The majority of nurse training schools have affiliated with institutions of higher learning. The affiliation offers increased learning opportunities for nursing students. Curriculum emphasis is on improving the educational atmosphere and less on service. As a result, the student nurse has a better academic background with which to move forward professionally.

Other area schools of nursing which have affiliated with academic institutions include Sibley Hospital (American University) and Freedmen's Hospital (Howard University). Also, Georgetown University, Catholic University, and Columbia Union College each have a collegiate school of nursing.

Washington Technical Institute and Federal City College have developed nurse training curricula to fill the gap created by the closing of Capital City School of Nursing. Students attend Washington Technical Institute for two years and receive an Associate Degree; those attending Federal City College engage in four year curriculum and receive an undergraduate degree. Students from both institutions receive their clinical experiences at D. C. General Hospital and this is expected to continue. It is hoped that those who pass the State Board examinations will become staff nurses at the Hospital.

The Capital City School of Nursing received its funding from the District of Columbia budget until September, 1970. For the remainder of the year, through the current and final year, the U.S. Department of Health, Education, and Welfare provided funds to enable the last class to complete requirements and graduate.

There have never been tuition or fees. However, students were required to purchase uniforms, books, and other educational supplies. Students have been receiving stipends amounting to $105 a month from which deductions are made for room, meals, and life and health insurance.

The Capital City School of Nursing has been accredited since 1950, when accreditation for nursing schools started. The accrediting agency is the National League of Nursing. Evaluations were made at regular intervals and included an analysis of faculty qualifications, entrance requirements, student attrition rate, curriculum, and State Board performance record.

During the phasing out period, the faculty at the School have retired, returned to college for additional academic work, moved to other locations and new positions, or have been reassigned to other D. C. General Hospital programs.

The physical facilities which have been utilized by the Capital City School of Nursing have been reprogrammed for utilization by Washington Technical Institute, Federal City College, Margaret Mary Washington School for Practical Nursing, and units of the Department of Human Resources.

During the years, bulletins, photos, curriculum, documents and other memorabilia have been saved. These were sent to the Mugar Memorial Library Nursing Archive, Boston University, for safekeeping. The Library has been designated by the American Nursing Association as the official depository for these materials. The documents are available to students and others interested in research.

For the past several years, Frederick H. Wescoe, Ed.D., R.N. has been the director of the Capital School of Nursing. He will continue his administrative functions in nursing at D. C. General Hospital.

RAYMOND L. STANDARD, M.D., M.P.H.
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book REVIEWS

Intestinal Transport of Electrolytes, Amino Acids, and Sugars. W. MCD. ARMSTRONG AND A.S. NUNN. Price, $21.50. Pp. 352. Springfield, Illinois: Charles C. Thomas, 1971.

This volume contains the proceedings of a Symposium held in September 1968. The papers are concerned with mechanisms of transport at the cellular and sub-cellular levels, and the list of contributors is one of marked distinction. The discussions cover a broad range of topics within the area defined by the title. In addition to work of well recognized significance, such as the effects of sodium on non-electrolyte transport or the specificity of the hexose and amino acid transport systems; the volume includes some papers on topics that have been less extensively investigated, for example the ontogeny of intestinal transport or the effects of mercury on intestinal transport processes. Even so, the lack of discussion of some important work prevents the volume from being considered as a reflection of the state of the art. For example, there is no discussion of the attempts to isolate hexose binding components of brush borders or the ion selectivity properties of membranes. An additional reservation must be expressed concerning the redundancy associated with the latent period of publication. This field is one of continuing activity and the volume contains little that can be considered either new or definitive. Thus the volume is of limited use to the specialist in this field. In contrast, the non-specialist interested in intestinal physiology may consider the volume to be a useful collection of milestones and signposts. Most of the papers are personal accounts of the authors' work and, if regarded as a forum for the presentation of the thinking of a number of well known workers in intestinal transport, the volume provides an indication of the type of problem that is of interest to this group.

The volume has been well produced with legible type and clear figures, although it is unfortunate that the publishers were unable to provide photographic illustrations for Trier's paper on intestinal morphology. The high price and limited scope will probably prevent the achievement of "best seller" status.

MICHAEL J. JACKSON Ph.D.

Genetic Disorders of the Endocrine Glands. DAVID L. RIMOIN, M.D., PH.D. AND R. NEIL SCHIMKE, M.D. Price, $32.50. Pp. 356 with illustrations. St. Louis: C.V. Mosby Company, 1971.

This volume presents a unique compilation of the large number of rare inherited disorders of the endocrine glands. The major contribution of the text is that it catalogues in a succinct manner each of the disorders and provides an appropriate bibliography to which the reader can refer for more detailed information. Since the majority of these disorders will probably never be seen by most physicians, the primary value of the book is as a reference text for the pediatrician or endocrinologist.

The book is divided into 9 chapters. The first chapter is concerned with the genetic aspects of clinical endocrinology and each of the subsequent chapters are devoted to one of the endocrine glands. The chapter presenting genetic disorders of the endocrine pancreas merits particular mention. This section contains a well written detailed discussion of the hereditary aspects of diabetes mellitus, a very confusing area which has been the subject of a great deal of controversy. The authors clearly present the reasons for the confusion, summarize the many published prevalance studies of diabetes and carefully evaluate the postulated modes of inheritance of this illness in an unbiased fashion. The chapter concludes with abnormal glucose tolerance including the hyperlipoproteinemias.

The major criticism of the text is that the authors do not conclude the treatment aspects of the various disorders beyond simple statements such as "treatment is supportive" or "treatment is surgical." This criticism is relatively minor since the primary focus of the book is to catalogue the clinical, hereditary and metabolic features of the various genetic disorders of the endocrine glands such that an appropriate diagnosis can be made. Development of a treatment program for these patients will require a greater understanding of the pathophysiology of the disorder than is possible within the framework of this text. In addition, the information required by the physician treating these patients is readily available through the extensive bibliography which accompanies each chapter.

In conclusion, the myriad of rare hereditary disorders of endocrinology usually present a diagnostic dilemma to the physician.

This text provides reference catalogue of these disorders to assist in making the correct diagnosis.

MICHAEL BALL, M.D.

The Medicine Men. LEONARD TUSHNET, M.D. Price, $7.95 Pp. 217. New York: St. Martin's Press, 1971.

This book "The Medicine Men" is directed to the layman. The subtitle is "The Myth of Quality Medical Care in America Today." Dr. Leonard Tushnet is a recently retired physician having practiced 40 years in New Jersey. The book, according to the publisher, is a full length analysis of the profession to which Dr. Tushnet has devoted most of his life. It is a relatively brief 217 pages. Its purpose is stated as helping the individual person become an intelligently active participant in his medical care. The entire tenor is one of "debunking" many sacrosanct beliefs of the American public. Any physician can profit considerably from reading the book inasmuch as what Dr. Tushnet says is true to a considerable extent. Many non-medically trained people will find the technical vocabulary in need of explanation. The physician reading it will, with proper reflection, find areas in which he can improve his techniques of caring for patients.

Unfortunately, many non-physicians may feel after reading the text carefully that physicians generally do not do the job that they should and that basically it is up to the patient, by questioning, verifying, et cetera, to assure themselves of proper medical care. I am sure conscientious physicians are always helped by the difficult patient and by the questioning intelligent patient. However, I would hope that the achievement of true quality medical care is a responsibility within the profession which may need to take many steps in order to assure this quality to the citizens of America.

A word of caution, some physicians may become somewhat angry as they start to read this book. I would think that holding back the anger and reflecting on what is said instead of perhaps being angered by the manner in which it is said will help make this book useful to all practicing physicians. I am not sure as to how useful it will be to the non-physician. In any case, however, I think being conversant with the book will be helpful in handling people who may question methods, diagnoses, laboratory examinations and the entire spectrum of medical care at this time.

MICHEL M. HEALY, M.D., P.A.

A Primer of Electrocardiography, 6th edition. GEORGE E. BURCH, M.D., F.A.C.P., F.A.C.C. AND TRAVIS WINSOR, M.D., F.A.C.P. Price, $7.75. Pp. 292 with 287 illustrations. Philadelphia: Lea & Febiger, 1972.

In reviewing the 6th edition of the classic monograph, one can hardly add anything to the authors' own preface. Drs. Burch & Winsor clearly point out that they still consider this to be a reference for "students and others with meager knowledge of electrocardiography." They further state that in a monograph of this type complex new concepts are not suitable for beginners and hence only the fundamental and classical principles are included. Finally, they readily admit that "the sixth edition was changed very little."

I had the opportunity to compare the book with a copy of the 2nd edition, published in 1949, and it is virtually identical, with few exceptions. For example, there are now several pages devoted to the principles of spatial vectorcardiography and a very brief few paragraphs concerning His Bundle electrograms (HBE). Unfortunately, there is no mention whatsoever of the very important and timely concepts of trifascicular block, the hemiblocks and bilateral bundle branch block.

In summary, although I feel that the authors continue to achieve their stated goals, as they have over the years, I believe that some of the aforementioned topics should be included with some basic discussion. The medical student, house officer and novice electrocardiographer are indeed encountering these terminologies daily.

LOWELL M. WEISS, M. D.

medical annals of the district of columbia

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Identification of Asthma. Ciba Foundation Study Group No. 38. RUTH PORTER AND JOAN BIRCH, Editors. Price, $8.25. Pp. 179 with illustrations. Edinburgh: Churchill Livingstone, 1971.

"The Identification of Asthma" is a detailed report of the Ciba Foundation Study Group No. 38 meeting in London, England, January 1971.

The title "Identification" (of Asthma) is aptly chosen. The panelists who met to "define" asthma (previous Ciba Foundation Groups have "defined" emphysema and chronic bronchitis) acknowledged that they had nothing new to add to the definition as written in 1684 by Thomas Willis: "An asthma is a most terrible disease . . . for there is scarce anything more sharp and terrible than the fits hereof . . . But as to evident causes they are many, and also of diverse sorts . . . Asthmatical persons can endure nothing violent, or unaccustomed: from excess of cold, or heat, from any vehement motion of body or mind, by any great change of Air, or of the year, or from the slightest errors about the things not natural, yea from a thousand other occasions they fall into fits of difficult breathing . . . Whatsoever therefore makes the blood to boyl, or raises it into an effervescence, as violent motion of the body or mind, excess of extern cold or heat, the drinking of Wine, Venery, yea sometimes meer heat of the Bed doth cause Asthmatical assaults to such as are predisposed."

The excellent explanation of the present status of asthma by the leaders in their respective fields of medicine pertaining to anatomy, pathology, physiology, pharmacology, chemistry and immunology is concisely and authoritatively presented. The fact that the term, asthma, is imprecise and subjective ". . .is unimportant as long as the aetiological mechanism is sought to enable rational treatment to be given."

The chapter, Clinical Review, is particularly informative to all physicians who treat asthmatic patients.

A panelist cleverly defined "Status Asthmaticus": "status asthmaticus is that condition in the asthmatic patient due to asthma which, if it doesn't frighten the doctor, ought to."

Although the panel did not "define" asthma they did "identify" it; and therefore, as stated by the chairman of the panel in his opening remarks, "bringing together research workers in different disciplines to discuss new developments and, sometimes, as in this case, old but unsolved problems" is of great value.

ELLIS, APRIL, M.D.

Manual of Tonography. ROBERT C. DREWS, M.D. Price, $13.75. Pp. 128 with 89 illustrations. St. Louis: C.V. Mosby Company, 1971.

In an introductory chapter on the history of tonography, the author sites a 1957 quotation from Dr. Morton Grant, the father of modern tonography, who said "tonography has not yet shown to be of practical value as an aid in the continuing management of glaucoma." While many ophthalmologists will agree with this statement even today, Dr. Drews sets out in his monograph to prove that tonography, for those who will use it, is an essential part of the modern ophthalmologist's armamentarium in treating glaucoma. The author has written a concise manual which leads the reader from theoretical considerations to the clinical application of tonography.

The author emphasizes the potential errors in tonography, inherent in the instrumentation. He also cites those due to lack of patient cooperation and the technical skill of the examiner. In spite of these errors however, he points out that careful tonography is the best method for reasonably estimating the facility of outflow in the living eye.

A complete discussion of tonographic equipment is considered in the 3rd chapter, noting the advantages and disadvantages as well as practical problems such as relative costs of the tonometers and recorders available commercially at this time. In this same chapter the author properly emphasizes that tonography is not a conclusive diagnostic tool in angle closure glaucoma but that the diagnosis depends on the gonioscopic evidence of angle occlusion at the time the pressure is elevated. He carefully emphasizes that gonioscopy is a *sine quo non* of diagnosis on dilation provocative tests for angle closure glaucoma.

The clinical application of tonography is carefully considered with many examples as to its usefulness and interpretation of the limitations in the individual case.

Despite the fact that this is a manual dealing with tonography, it is disappointing to note that no mention is made of optic nerve damage or visual field change in correlation with tonographic evidence of glaucoma. Nevertheless, this manual provides a useful guide to the clinician interested in tonography and should be included in any library where tonographic facilities exist. It is not however, recommended as a "must" for the library of all ophthalmologists.

MELVIN G. ALPER, M.D.

Handbook of Pediatrics, 9th edition. HENRY K. SILVER, M.D., HENRY C. KEMPE, M.D., AND HENRY B. BRUYN, M.D., Editors. Price, $6.50. Pp. 774, with illustrations. Los Altos, California: Lange Medical Publications, 1971.

In this pocket-sized volume, the entire range of pediatric disorders is covered in outline form. The style is crisp and the presentations concise. Many tables of differential diagnosis and tabular classifications of related disorders are included. Only a few references are cited but the work is not intended to be a text book.

A useful appendix includes drug dosages, technical hints, laboratory aids, normal value tables, and the like.

For the student and house officer, this is a useful *vade mecum*. For the practitioner, it will serve as a memory jogger. I recommend it.

ALLAN B. COLEMAN, M.D.

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Warnings: Age, weight, dosage, duration of therapy, existence of concomitant diseases, and concurrent potent chemotherapy affect incidence of toxic reactions. Carefully instruct and observe the individual patient, especially the aging (forty years and over) who have increased susceptibility to the toxicity of the drug. Use lowest effective dosage. Weigh initially unpredictable benefits against potential risk of severe, even fatal, reactions. The disease condition itself is unaltered by the drug. Use with caution in first trimester of pregnancy and in nursing mothers. Drug may appear in cord blood and breast milk. Serious, even fatal, blood dyscrasias, including aplastic anemia, may occur suddenly despite regular hemograms, and may become manifest days or weeks after cessation of drug. Any significant change in total white count, relative decrease in granulocytes, appearance of immature forms, or fall in hematocrit should signal immediate cessation of therapy and complete hematologic investigation. Unexplained bleeding involving CNS, adrenals, and G.I. tract has occurred. The drug may potentiate action of insulin, sulfonylurea, and sulfonamide-type agents. Carefully observe patients taking these agents. Nontoxic and toxic goiters and myxedema have been reported (the drug reduces iodine uptake by the thyroid). Blurred vision can be a significant toxic symptom worthy of a complete ophthalmological examination. Swelling of ankles or face in patients under sixty may be prevented by reducing dosage. If edema occurs in patients over sixty, discontinue drug.
Precautions: The following should be accomplished at regular intervals: Careful detailed history for disease being treated and detection of earliest signs of adverse reactions; complete physical examination including check of patient's weight; complete weekly (especially for the aging) or an every two week blood check; pertinent laboratory studies. Caution patients about participating in activity requiring alertness and coordination, as driving a car, etc. Cases of leukemia have been reported in patients with a history of short- and long-term therapy. The majority of these patients were over forty. Remember that arthritic-type pains can be the presenting symptom of leukemia.
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# of GENERAL INTEREST

A Silver Anniversary

1972 marks twenty-five years of partnership for the control of cancer between the D.C. Division of the American Cancer Society and the Medical Society of the District of Columbia. Throughout this past quarter century physicians and dentists have been and continue to be active participants in cancer control programs in the nation and especially in the Washington area. The inception of the D.C. Division in 1947 was fostered by eminent members of Washington's Medical Society and in the ensuing twenty-five years many of the Society's members have actively served the Division in leadership capacities.

The first outstanding achievement of this partnership came in 1947 with the development of criteria for cancer detection centers and diagnostic treatment clinics. That same year the D.C. Division awarded its first grant to instruct visiting nurses in the in-home care of cancer patients. This was just the beginning, for the progress and accomplishments have been continuous.

The first Cancer Registry in D.C. was established in 1949 and in the same year the first Clinical Fellowship Grant for cancer surgery was awarded to an area physician, Dr. Jack E. White, at the Memorial Hospital for Cancer and Allied Diseases, N.Y.

Also in 1949, a one-day cancer symposium was held at the Annual Assembly of the D.C. Medical Society and each year following, there has been an exhibit presented by the D.C. Division. Another accomplishment was the organization of the Cured Cancer Club. Then came the concern of Otolaryngologists, who were instrumental in organizing the Lost Chord Club for psychological adjustment of the laryngectomized patient.

Congressional action spurred by pressure from the Medical Society made cancer a mandatory reportable disease in Washington—July 27, 1951.

A cooperative innovation came in the form of a telephone service conducted by physicians to give "Answers to Cancer." This proved so popular in the community that it became a continuous program. Recently, the public responded with 750 calls in a 48-hour period to hear a smoking and health message.

Another popular program is the D.C. Division's Professional Education Program which includes an accredited postgraduate course for practicing dentists, a lecture series on cancer diagnosis and treatment for the general practitioner, and the St. George Society which helps to promote knowledge of cancer among medical students.

It is estimated that D.C. will have 3,100 new cases of cancer and 1,600 deaths from cancer in 1972. With these statistics it becomes clear that the community will have an even greater need for programs, information and services.

The American Cancer Society, D.C. Division, has in existence many fine service oriented programs. A counseling service and a therapeutic communications service dealing with information, loneliness, fear, anxiety and various other problems of the family and patient are provided. Community Service Programs also include rehabilitation service and support to create new cancer registries.

Rehabilitation services aim to assist the patient in his/her return to a socially useful and personally significant life. The Laryngectomy Program provides a rehabilitation film, laryngectomy kit (booklets, bibs and emergency cards, etc.) speech therapy, a visitors program and the Lost Chord Club. Reach to Recovery, a rehabilitation program for mastectomy patients, offers help to the new patient from women who have experienced the same anxieties, fears, and mental and physical pain resulting from radical breast surgery. All services are provided free and patients must be referred by their attending physician. Aids such as colostomy and ileostomy appliances, breast prostheses and maxillofacial prostheses are provided by the D.C. Division for the medically indigent patient's physical and social restoration.

In 1969, the idea for a Special Case-Finding Project was developed. The purpose was to locate cancer patients as soon after diagnosis as possible and offer assistance through contacts with physicians, dentists, the cancer registries, and the pathology departments of area hospitals. This project is aimed at avoiding financial disaster and the anxiety and frustration of cancer patients and their families which so often accompany this diagnosis. No patient or family member is contacted or approached without the expressed consent and authorization of the attending physician. The Special Case-Finding Project has received the endorsement of the Executive Board of the Medical Society of D.C., the Section on Pathology and Laboratory Medicine of the Medical Society of D.C. and the Medico-Chirurgical Society.

Only through our continuing cooperative efforts can our fight against cancer be effective.

NEW MEMBERS

Active

SIRUS AMIRI, M.D. Office: 5401 Western Avenue. Born in Teheran, Iran. Medical degree: Teheran University, 1956. Specialty: Pediatrics and Pediatric Cardiology.

RICHARD B. D'ALLESSIO, M.D. Office: 5225 Connecticut Avenue, N.W. Born in Pittsburgh, Pennsylvania. Medical degree: Temple Medical School, 1964. Specialty: Pediatrics and Pediatric Allergy.

JOHN W. DOWNING, M.D. Office: Freedmen's Hospital. Born in Phoebus, Virginia. Medical degree: Meharry Medical College, 1961. Specialty: Pediatrics and Pediatric Cardiology.

STEPHAN P. GINSBERG, M.D. Office: George Washington University Hospital. Born in Columbus, Ohio. Medical degree: University of California (San Francisco), 1966. Specialty: Ophthalmology.

ARTHUR H. HOYTE, M.D. Office: 3700 Reservoir Road. Born in Boston, Massachusetts. Medical degree: Columbia College of Physicians and Surgeons, 1964. Specialty: Obstetrics–Gynecology.

HAMID MONTAKHAB, M.D. Office: 2121 Pennsylvania Avenue, N.W. Born in Shiraz, Iran. Medical degree: University of Geneva, 1962. Specialty: Internal Medicine.

ROBERTO N. SUNGA, M.D. Office: 2150 Pennsylvania Avenue, N.W. Born in Manila, Philippines. Medical degree: University of the Philippines, 1958. Specialty: Ophthalmology.

ELIZA TAYLOR, M.D. Office: 2101 16th Street, N.W. Born in Columbia, Virginia. Medical degree: Howard University, 1967. Specialty: Internal Medicine.

Associate

BARRY A. COHEN, M.D. Office: 510 N Street, S.W. Born in New York, New York. Medical degree: Cornell Medical College, 1965. Specialty: Psychiatry.

ROBERT KATZ, M.D. Office: 1109 Spring Street, Silver Spring, Maryland. Born in Brooklyn, New York. Medical degree: George Washington University, 1961. Specialty: Dermatology.

Resident-Intern

RALPH W. MOORE, M.D. Office: Georgetown University Medical Center. Born in Springfield, Massachusetts. Medical degree: Georgetown University, 1969. Specialty: Psychiatry.

EUGENE W. WILLIAMS, M.D. Office: Freedmen's Hospital. Born in Washington, D.C. Medical degree: Howard University, 1970. Specialty: Obstetrics-Gynecology.

Student

GEORGE WASHINGTON UNIVERSITY. *Freshman:* Gregory Berlin.

Changes in Membership

Transfers

Active to Associate: Margaret W. Bridwell, M.D., Bernard J. Hillig, M.D., James T. Waters, M.D., Joseph I. Wollman, M.D.

Associate to Active: Victor A. Khayat, M.D.

Active to Special: Henry H. Canton, M.D., Frank D. Costenbader, M.D.

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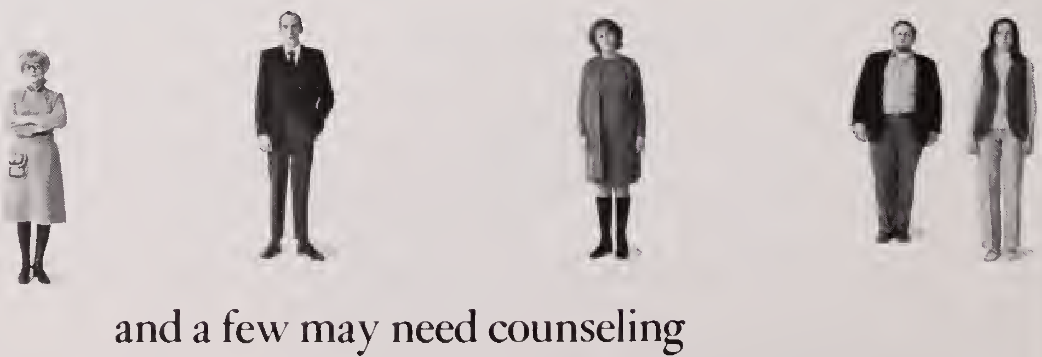

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Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence. In pregnancy, lactation or women of childbearing age, weigh potential benefit against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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obituaries

Don S. Knowlton, M.D.
1893–1972

Dr. Don S. Knowlton, who practiced otolaryngology in the Washington area from 1927 until his retirement in 1965, died April 8, 1972. He resided at 1805 Hoban Road, N.W.

He was born in Fairfield, Maine and was graduated from Yale University with a Bachelor of Science degree. He received his medical degree from Tufts University and interned at Cambridge Hospital in Massachusetts and New Haven Hospital in Connecticut. He served his residency at University Hospital in Syracuse, New York. In addition to maintaining offices in the Columbia Medical Building, Dr. Knowlton was an Associate Professor of Otolaryngology at Georgetown University Medical School. He was also a consultant at George Washington University Hospital and at Doctors Hospital, as well as the old Mount Alto Veterans Administration Hospital.

In World War I Dr. Knowlton served with the American Expeditionary Forces in France and in the 1920's with the Organized Reserve Corps. He was commissioned in the Naval Reserve in 1930. During World War II, he received a Legion of Merit for outstanding service as Executive Officer of the first medical battalion during the seizure and occupation of Guadalcanal. Later he received a Gold Star in lieu of a second Legion of Merit for duty as division surgeon of a Marine infantry division on Okinawa. In addition, he was awarded the Purple Heart for combat in the Pacific. He remained a reserve naval officer until his retirement as a Rear Admiral in 1950.

Dr. Knowlton was a Life member of the Medical Society of the District of Columbia.

He is survived by his wife, Mary Young Knowlton, of the home address.

Gerhart J. Gordon, M.D.
1903–1972

Dr. Gerhart J. Gordon, a psychiatrist with the D.C. government since 1950, died April 8, 1972.

Born in Berlin, Germany, he received his medical degree from the University of Berlin and took his postgraduate training at the Psychiatric Clinic of the University of Paris. He interned at the Municipal Hospitals of Berlin-Britz and Berlin-Moabit, and served his residency at the Home for Brain-Injured Veterans in Frankfurt. From 1934 to 1950 he held a position on the staff of the Delaware State Hospital. In 1950 he became chief medical officer in the department of psychiatry at Gallinger Hospital, which is now D.C. General Hospital. In 1965 he was appointed supervisory medical officer of the psychiatry, medical care and hospital treatment division at D.C. General Hospital, and in 1968 he was named medical officer of psychiatry for the Area B Community Health Center under the D.C. Department of Human Resources.

In addition to his membership in the Medical Society of the District of Columbia, Dr. Gordon was a member of the American Psychiatric Association and the American Medical Association.

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Richmond C.A. Brooks, M.D.
1899–1972

Dr. Richmond C. A. Brooks, M.D., who practiced otolaryngology in the Washington area for 50 years, died April 15, 1972. He resided at 6708 Hillandale Road, Chevy Chase, Maryland.

A native of Washington, D.C., he received his Bachelor of Science and medical degrees from Georgetown University. He interned at Emergency and Providence Hospitals and served his residency at the Episcopal Eye, Ear, Nose and Throat Hospital. He most recently held a staff appointment at Surburban Hospital in Bethesda.

Dr. Brooks served with the Army in World War I and as consulting otolaryngologist at Fort Myer, Virginia, during World War II. He was decorated with the Three Stars of the Latvian government.

A Life member of the Medical Society of the District of Columbia, he was also a member of the Montgomery County Medical Society and a fellow of the American Academy of Ophthalmology and Otolaryngology.

Dr. Brooks is survived by his wife, Rosa, of the home; a son, Richmond, Jr., of Bethesda; a daughter, Mrs. Charles C. Hall, of Kensington, and nine grandchildren.

Caroline Jackson, M.D.
1908–1972

Dr. Caroline Jackson, a retired Washington area physician, died April 8, 1972. She resided at 2032 Belmont Road, N.W.

Born in New York, New York, she received a Bachelor of Arts degree from the University of California and a Master of Arts degree from Columbia University. She received her medical degree from Johns Hopkins University, where she served her internship and residency. She was an Assistant Clinical Professor of Obstetrics and Gynecology at George Washington University Medical School and maintained a practice in obstetrics and gynecology until 1969, when she became semi-retired.

A member of the Medical Society of the District of Columbia since 1939, Dr. Jackson was Third Vice President 1948–1949 and served on the Medical Bureau Committee for 18 years. She held a Life membership in the American College of Obstetrics and Gynecology and Washington Gynecological Society.

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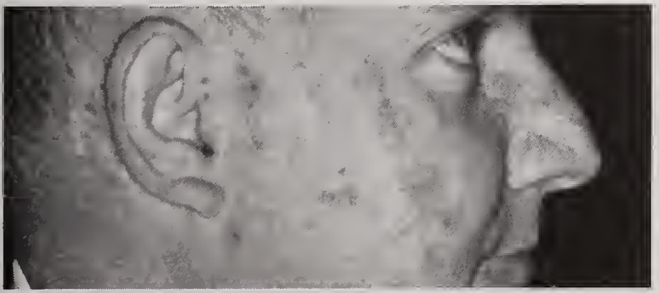

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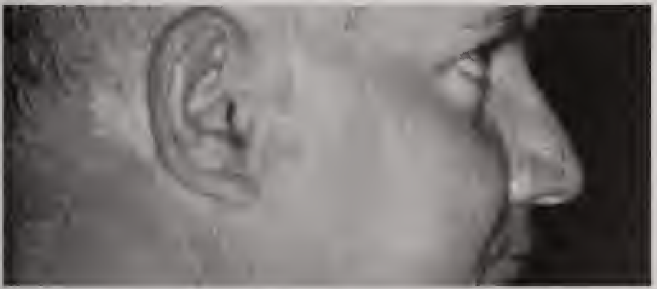

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*Data on file, Hoffmann-La Roche Inc., Nutley, N.J.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Multiple actinic or solar keratoses.

Contraindications: Patients with known hypersensitivity to any of its components.

Warnings: If occlusive dressing used, may increase inflammatory reactions in adjacent normal skin. Avoid prolonged exposure to ultraviolet rays. Safe use in pregnancy not established.

Precautions: If applied with fingers, wash hands immediately. Apply with care near eyes, nose and mouth. Lesions failing to respond or recurring should be biopsied.

Adverse Reactions: Local — pain, pruritus, hyperpigmentation and burning at application site most frequent; also dermatitis, scarring, soreness and tenderness. Also reported — insomnia, stomatitis, suppuration, scaling, swelling, irritability, medicinal taste, photosensitivity, lacrimation, leukocytosis, thrombocytopenia, toxic granulation and eosinophilia.

Dosage and Administration: Apply sufficient quantity to cover lesion twice daily with nonmetal applicator or suitable glove. Usual duration of therapy is 2 to 4 weeks.

How Supplied: Solution, 10-ml drop dispensers — containing 2% or 5% fluorouracil on a weight/weight basis, compounded with propylene glycol, tris(hydroxymethyl)aminomethane, hydroxypropyl cellulose, parabens (methyl and propyl) and disodium edetate.

Cream, 25-Gm tubes — containing 5% fluorouracil in a vanishing cream base consisting of white petrolatum, stearyl alcohol, propylene glycol, polysorbate 60 and parabens (methyl and propyl).

This patient's lesions were resolved with

Efudex®
(fluorouracil)
5% cream/solution
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Division of Hoffmann-La Roche Inc.
Nutley, N.J. 07110

Pre-Sate® (chlorphentermine hydrochloride)

Caution: Federal law prohibits dispensing without prescription.

Indications

Pre-Sate (chlorphentermine hydrochloride) is indicated in exogenous obesity, as a short term (i.e. several weeks) adjunct in a regimen of weight reduction based upon caloric restriction.

Contraindications

Glaucoma, hyperthyroidism, pheochromocytoma, hypersensitivity to sympathomimetic amines, and agitated states. Pre-Sate (chlorphentermine hydrochloride) is also contraindicated in patients with a history of drug abuse or symptomatic cardiovascular disease of the following types: advanced arteriosclerosis, severe coronary artery disease, moderate to severe hypertension, or cardiac conduction abnormalities with danger of arrhythmias. The drug is also contraindicated during or within 14 days following administration of monamine oxidase inhibitors, since hypertensive crises may result.

Warnings

When weight loss is unsatisfactory the recommended dosage should not be increased in an attempt to obtain increased anorexigenic effect, discontinue the drug. Tolerance to the anorectic effect may develop. Drowsiness or stimulation may occur and may impair ability to engage in potentially hazardous activities such as operating machinery, driving a motor vehicle, or performing tasks requiring precision work or critical judgment. Therefore, such patients should be cautioned accordingly. Caution must be exercised if Pre-Sate (chlorphentermine hydrochloride) is used concomitantly with other central nervous system stimulants. There have been reports of pulmonary hypertension in patients who received related drugs.

Drug Dependence Drugs of this type have a potential for abuse. Patients have been known to increase the intake of drugs of this type to many times the dosages recommended. In long-term controlled studies with the high dosages of Pre-Sate, abrupt cessation did not result in symptoms of withdrawal.

Usage In Pregnancy The safety of Pre-Sate (chlorphentermine hydrochloride) in human pregnancy has not yet been clearly established The use of anorectic agents by women who are or who may become pregnant, and especially those in the first trimester of pregnancy, requires that the potential benefit be weighed against the possible hazard to mother and child. Use of the drug during lactation is not recommended. Mammalian reproductive and teratogenic studies with high multiples of the human dose have been negative.

Usage In Children Not recommended for use in children under 12 years of age.

Precautions

In patients with diabetes mellitus there may be alteration of insulin requirements due to dietary restrictions and weight loss. Pre-Sate (chlorphentermine hydrochloride) should be used with caution when obesity complicates the management of patients with mild to moderate cardiovascular disease or diabetes mellitus, and only when dietary restriction alone has been unsuccessful in achieving desired weight reduction. In prescribing this drug for obese patients in whom it is undesirable to introduce CNS stimulation or pressor effect, the physician should be alert to the individual who may be overly sensitive to this drug. Psychologic disturbances have been reported in patients who concomitantly receive an anorectic agent and a restrictive dietary regimen.

Adverse Reactions

Central Nervous System: When CNS side effects occur, they are most often manifested as drowsiness or sedation or overstimulation and restlessness. Insomnia, dizziness, headache, euphoria, dysphoria, and tremor may also occur. Psychotic episodes, although rare, have been noted even at recommended doses. **Cardiovascular:** tachycardia, palpitation, elevation of blood pressure. **Gastrointestinal:** nausea and vomiting, diarrhea, unpleasant taste, constipation. **Endocrine:** changes in libido, impotence. **Autonomic:** dryness of mouth, sweating, mydriasis. **Allergic:** urticaria. **Genitourinary:** diuresis and, rarely, difficulty in initiating micturition. **Others:** Paresthesias, sural spasms.

Dosage and Administration

The recommended adult daily dose of Pre-Sate (chlorphentermine hydrochloride) is one tablet (equivalent to 65 mg chlorphentermine base) taken after the first meal of the day. Use in children under 12 not recommended.

Overdosage

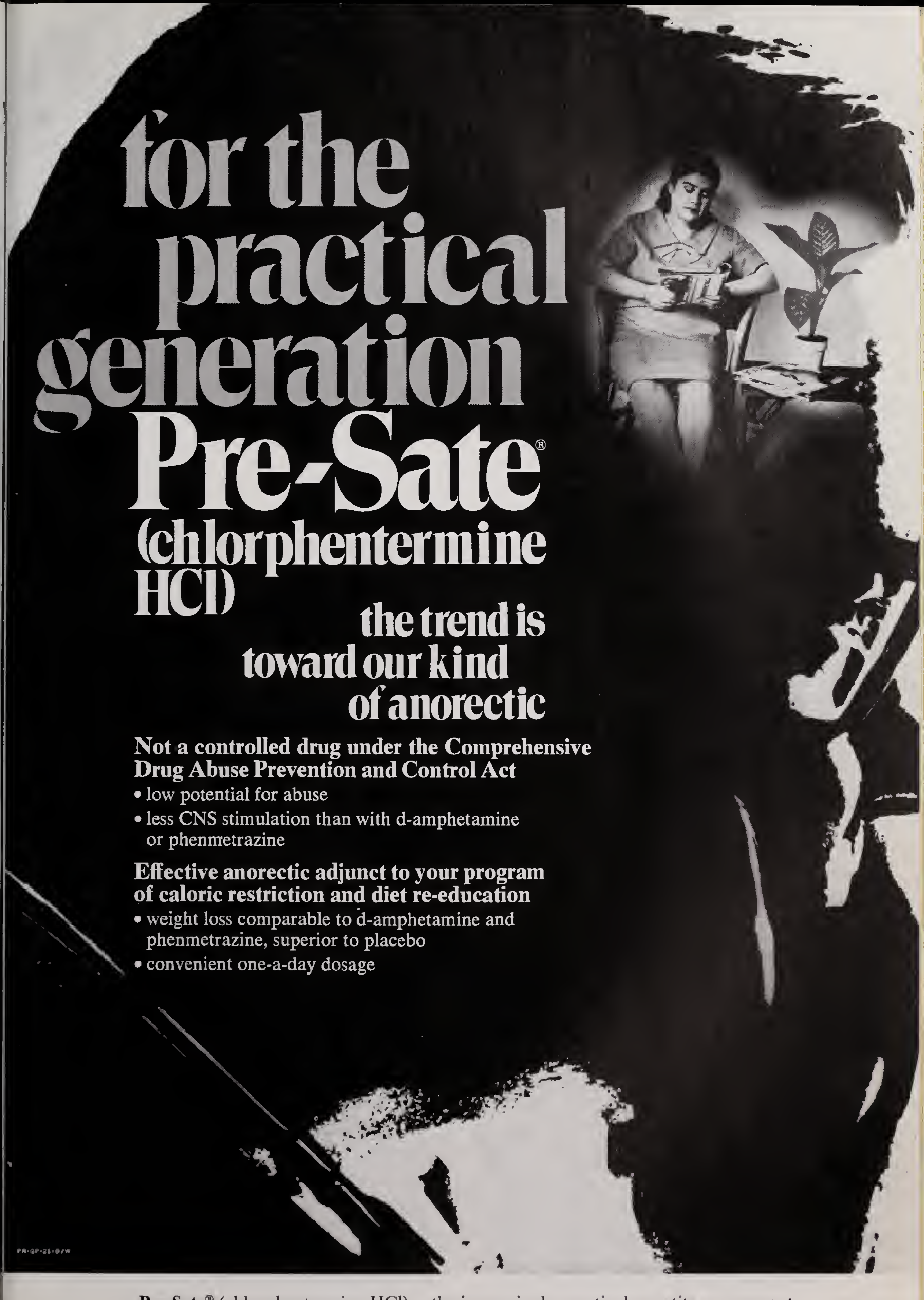
Manifestations: Restlessness, confusion, assaultiveness, hallucinations, panic states, and hyperpyrexia may be manifestations of acute intoxication with anorectic agents. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension, or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Fatal poisoning usually terminates in convulsions and coma.

Management: Management of acute intoxication with sympathomimetic amines is largely symptomatic and supportive and often includes sedation with a barbiturate. If hypertension is marked, the use of a nitrate or rapidly acting alpha-receptor blocking agent should be considered. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendations in this regard.

How Supplied

Each Pre-Sate (chlorphentermine hydrochloride) tablet contains the equivalent of 65 mg chlorphentermine base; bottles of 100 and 1000 tablets.

Full information is available on request.



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Effective anorectic adjunct to your program of caloric restriction and diet re-education

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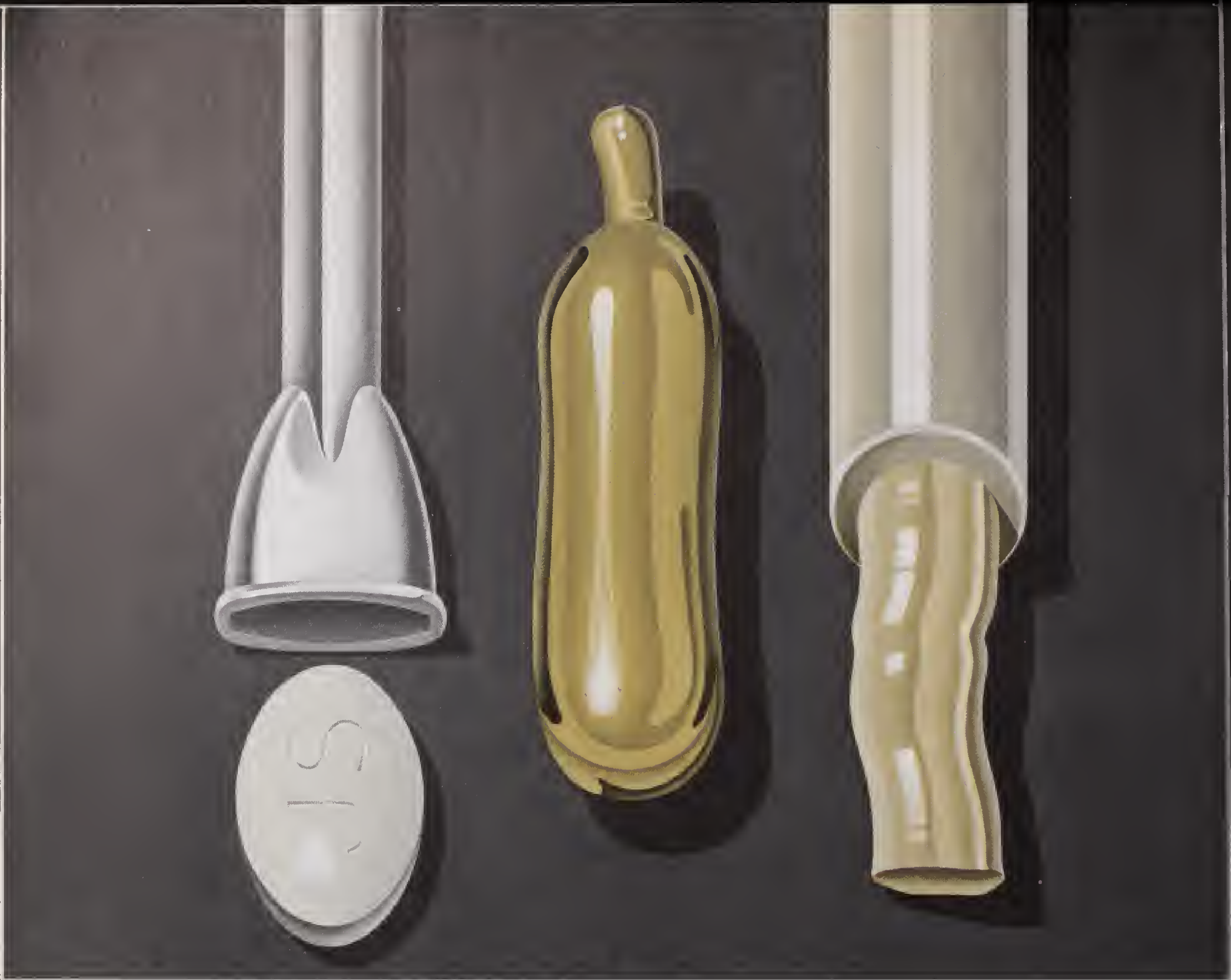

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(candicidin)

Description: Candeptin (candicidin) Vaginal Ointment contains a dispersion of candicidin powder equivalent to 0.6 mg. per gm. or 0.06% Candicidin activity in U.S.P. petrolatum. 3 mg. of Candicidin is contained in 5 gm. of ointment or one applicatorful. Candeptin Vaginal Tablets contain Candicidin powder equivalent to 3 mg. (0.3%) Candicidin activity dispersed in starch, lactose and magnesium stearate. Candeptin Vagelettes Vaginal Capsules contain 3 mg. of Candicidin activity dispersed in 5 gm. U.S.P. petrolatum.
Action: Candeptin Vaginal Ointment, Vaginal Tablets, and Vagelettes Vaginal Capsules possess anti-monilial activity.
Indications: Vaginitis due to Candida albicans and other Candida species.
Contraindications: Contraindicated for patients known to be sensitive to any of its components. During pregnancy manual Tablet or Vagelettes Capsule insertion may be preferred since the use of the ointment applicator or tablet inserter may be contraindicated.
Caution: During treatment it is recommended that the patient refrain from sexual intercourse or the husband wear a condom to avoid re-infection.
Adverse Reaction: Clinical reports of sensitization or temporary irritation with Candeptin Vaginal Ointment, Vaginal Tablets or Vagelettes Vaginal Capsules have been extremely rare.
Dosage: One vaginal applicatorful of Candeptin Ointment or one Vaginal Tablet or one Vagelettes Vaginal Capsule is inserted high in the vagina twice a day, in the morning and at bedtime, for 14 days. Treatment may be repeated if symptoms persist or reappear.
Available Dosage Forms: Candeptin Vaginal Ointment is supplied in 75 gm. tubes with applicator (14-day regimen requires 2 tubes). Candeptin Vaginal Tablets are packaged in boxes of 28, in foil with inserter—enough for a full course of treatment. Candeptin Vagelettes Vaginal Capsules are packaged in boxes of 14 (14-day regimen requires 2 boxes.)

Store under refrigeration to insure full potency.

Federal law prohibits dispensing without prescription.

References: **1.** Olsen, J.R., Journal-Lancet *85* 287 (July) 1965. **2.** Giorlando, S.W.: Ob/Gyn Dig. *13*:32 (Sept.) 1971. **3.** Decker, A.: Case Reports on File, Medical Department, Julius Schmid **4.** Giorlando, S.W., Torres, J.F., and Muscillo, G., Am. J. Obst. & Gynec. *90*: 370 (Oct. 1) 1964. **5.** Lechevalier, H Antibiotics Annual 1959-1960. New York, Antibiotica Inc., 1960. pp. 614-618. **6.** Friedel, H J Maryland M.J., *15*:36 (Feb.) 1966.

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Reference Example

1. GOODMAN, J., ET AL: Bacterial endocarditis as a possible complication of chronic hemodialysis. New Eng. J. Med., 1969, 280, 876.

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a new outlook in chronic pain

of moderate to severe intensity

Though Talwin® can be compared to codeine in analgesic efficacy, it is not a narcotic. So patients receiving Talwin for prolonged periods face fewer of the consequences you've come to expect with narcotic analgesics. And that, in the long run, can mean a better outlook for your chronic-pain patient.

Talwin Tablets are:

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- **Not subject to narcotic controls:** convenient to prescribe—day or night—even by phone.
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50 mg. Tablets

Talwin®

brand of pentazocine (as hydrochloride)

the long-range analgesic

a new outlook in chronic pain

of moderate to severe intensity

Contraindications: Talwin, brand of pentazocine (as hydrochloride), should not be administered to patients who are hypersensitive to it.

Warnings: *Head Injury and Increased Intracranial Pressure.* The respiratory depressant effects of Talwin and its potential for elevating cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions, or a preexisting increase in intracranial pressure. Furthermore, Talwin can produce effects which may obscure the clinical course of patients with head injuries. In such patients, Talwin must be used with extreme caution and only if its use is deemed essential.

Usage in Pregnancy. Safe use of Talwin during pregnancy (other than labor) has not been established. Animal reproduction studies have not demonstrated teratogenic or embryotoxic effects. However, Talwin should be administered to pregnant patients (other than labor) only when, in the judgment of the physician, the potential benefits outweigh the possible hazards. Patients receiving Talwin during labor have experienced no adverse effects other than those that occur with commonly used analgesics. Talwin should be used with caution in women delivering premature infants.

Drug Dependence. There have been instances of psychological and physical dependence on parenteral Talwin in patients with a history of drug abuse and, rarely, in patients without such a history. Abrupt discontinuance following the extended use of parenteral Talwin has resulted in withdrawal symptoms. There have been a few reports of dependence and of withdrawal symptoms with orally administered Talwin. Patients with a history of drug dependence should be under close supervision while receiving Talwin orally.

In prescribing Talwin for chronic use, the physician should take precautions to avoid increases in dose by the patient and to prevent the use of the drug in anticipation of pain rather than for the relief of pain.

Acute CNS Manifestations. Patients receiving therapeutic doses of Talwin have experienced, in rare instances, hallucinations (usually visual), disorientation, and confusion which have cleared spontaneously within a period of hours. The mechanism of this reaction is not known. Such patients should be very closely observed and vital signs checked. If the drug is reinstituted it should be done with caution since the acute CNS manifestations may recur.

Usage in Children. Because clinical experience in children under 12 years of age is limited, administration of Talwin in this age group is not recommended.

Ambulatory Patients. Since sedation, dizziness, and occasional euphoria have been noted, ambulatory patients should be warned not to operate machinery, drive cars, or unnecessarily expose themselves to hazards.

Precautions: *Certain Respiratory Conditions.* Although respiratory depression has rarely been reported after oral administration of Talwin, the drug should be administered with caution to patients with respiratory depression from any cause, severe bronchial asthma and other obstructive respiratory conditions, or cyanosis.

Impaired Renal or Hepatic Function. Decreased metabolism of the drug by the liver in extensive liver disease may predispose to accentuation of side effects. Although laboratory tests have not indicated that Talwin causes or increases renal or hepatic impairment, the drug should be administered with caution to patients with such impairment.

Myocardial Infarction. As with all drugs, Talwin should be used with caution in patients with myocardial infarction who have nausea or vomiting.

Biliary Surgery. Until further experience is gained with the effects of Talwin on the sphincter of Oddi, the drug should be used wit caution in patients about to undergo surgery of the biliary trac *Patients Receiving Narcotics.* Talwin is a mild narcotic antagonis Some patients previously receiving narcotics have experienced mil withdrawal symptoms after receiving Talwin.

CNS Effect. Caution should be used when Talwin is administere to patients prone to seizures; seizures have occurred in a few suc patients in association with the use of Talwin although no cause an effect relationship has been established.

Adverse Reactions: Reactions reported after oral administratio of Talwin include *gastrointestinal:* nausea, vomiting; infrequentl constipation; and rarely abdominal distress, anorexia, diarrhe *CNS effects:* dizziness, lightheadedness, sedation, euphoria, hea ache; infrequently weakness, disturbed dreams, insomnia, syncop visual blurring and focusing difficulty, hallucinations (see *Acu CNS Manifestations* under WARNINGS); and rarely tremor, irr tability, excitement, tinnitus. *Autonomic:* sweating; infrequentl flushing; and rarely chills. *Allergic:* infrequently rash; and rarel urticaria, edema of the face. *Cardiovascular:* infrequently decreas in blood pressure, tachycardia. *Other:* rarely respiratory depressio urinary retention.

Dosage and Administration: *Adults.* The usual initial adult dose 1 tablet (50 mg.) every three or four hours. This may be increase to 2 tablets (100 mg.) when needed. Total daily dosage should no exceed 600 mg.

When antiinflammatory or antipyretic effects are desired in add tion to analgesia, aspirin can be administered concomitantly wit Talwin.

Children Under 12 Years of Age. Since clinical experience in chi dren under 12 years of age is limited, administration of Talwin i this age group is not recommended.

Duration of Therapy. Patients with chronic pain who have receive Talwin orally for prolonged periods have not experienced wit drawal symptoms even when administration was abruptly disco tinued (see WARNINGS). No tolerance to the analgesic effect h been observed. Laboratory tests of blood and urine and of liver an kidney function have revealed no significant abnormalities afte prolonged administration of Talwin.

Overdosage: *Manifestations.* Clinical experience with Talwin ove dosage has been insufficient to define the signs of this condition.

Treatment. Oxygen, intravenous fluids, vasopressors, and othe supportive measures should be employed as indicated. Assisted c controlled ventilation should also be considered. Although nalo phine and levallorphan are *not* effective antidotes for respirator depression due to overdosage or unusual sensitivity to Talwin, pa enteral naloxone (Narcan®, available through Endo Laboratories) i a specific and effective antagonist. If naloxone is not available, pa enteral administration of the analeptic, methylphenidate (Ritalin® may be of value if respiratory depression occurs.

Talwin is not subject to narcotic controls.

How Supplied: Tablets, peach color, scored. Each tablet contai Talwin (brand of pentazocine) as hydrochloride equivalent to 50 m base. Bottles of 100.

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Antianxiety effectiveness: Demonstrated in a broad range of psychologic and physical dysfunctions; indicated when reassurance and counseling are not enough and until, in the physician's judgment, anxiety has been reduced to tolerable, appropriate levels.

Effect on mental acuity: Usually minimal on proper maintenance dosage. (See Warnings in summary of prescribing information.)

Concomitant use: Is used as adjunctive antianxiety therapy concomitantly with certain specific medications of other classes of drugs, such as cardiac glycosides, antihypertensive agents, diuretics and vasodilators.

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5-mg, 10-mg, 25-mg capsules
up to 100 mg daily in
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Before prescribing, please consult complete product information, a summary of which follows:
Indications: Indicated when anxiety, tension and apprehension are significant components of the clinical profile.
Contraindications: Patients with known hypersensitivity to the drug.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported. Use of any drug in pregnancy, lactation, or in women of childbearing age requires that its potential benefits be weighed against its possible hazards.
Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.
Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.
Supplied: Librium capsules containing 5 mg, 10 mg or 25 mg chlordiazepoxide HCl. Libritabs® tablets containing 5 mg, 10 mg or 25 mg chlordiazepoxide.

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PRESIDENT'S MESSAGE

Darrell C. Crain, M.D.

LOVE NEEDS CARE

And according to the physicians in the Community Health and Hospital Administration of the D.C. Department of Human Resources, love is not getting the care it needs - namely, protection from venereal disease. As a consequence, we share with the rest of the world the results of the rising incidence of syphilis and gonorrhea. The actual figures seem almost unbelievable: over 11,000 cases of gonorrhea actually reported in 1971 with estimates running as high as nearly 100,000; over 600 cases of infectious syphilis reported with a total estimate of 25 to 30 per cent above this figure. Considering that we are dealing with diseases of known cause, readily treatable, and easily prevented, one is tempted to shake his head and say, "How can this be?" Sociologists, psychiatrists, sexologists and epidemiologists may present varying explanations. But all agree that the problem is a universal one and is increasing. Your medical society is therefore going to join with others in the metropolitan area and with the various public health officials in a massive campaign designed to focus attention on the situation and lay the groundwork for bringing these scourges under control. The slogan adopted forms the title of this editorial.

Dr. James A. Curtin, chairman of our Committee on Communicable Disease, is over-all chairman of the drive. Assisting him are Drs. Charles R. Hayman and John R. Pate of the Community Health and Hospital Administration of the D.C. Department of Human Resources. Serving on the Policy and Steering Committee are lay as well as medical representatives of Montgomery, Prince Georges, Arlington, Fairfax and Prince William Counties.

Current plans call for an intense educational drive culminating in VD Week, October 8. Our Committee on Community Health under Dr. Erman W. Edgecombe has voted to make that week our annual "Community Health Week"; our Committee on Medicine and Religion under Dr. Angelo D'Agostino has already presented a symposium at one of our churches, developed by the Reverend Joseph Tartt of the Grace Church in Northwest Washington; our Committee on Public Relations under Dr. John M. Keshishian will be furnishing speakers throughout the area, and with public relations officer, Mr. Lou Brott, will be working to publicize the facts throughout the area.

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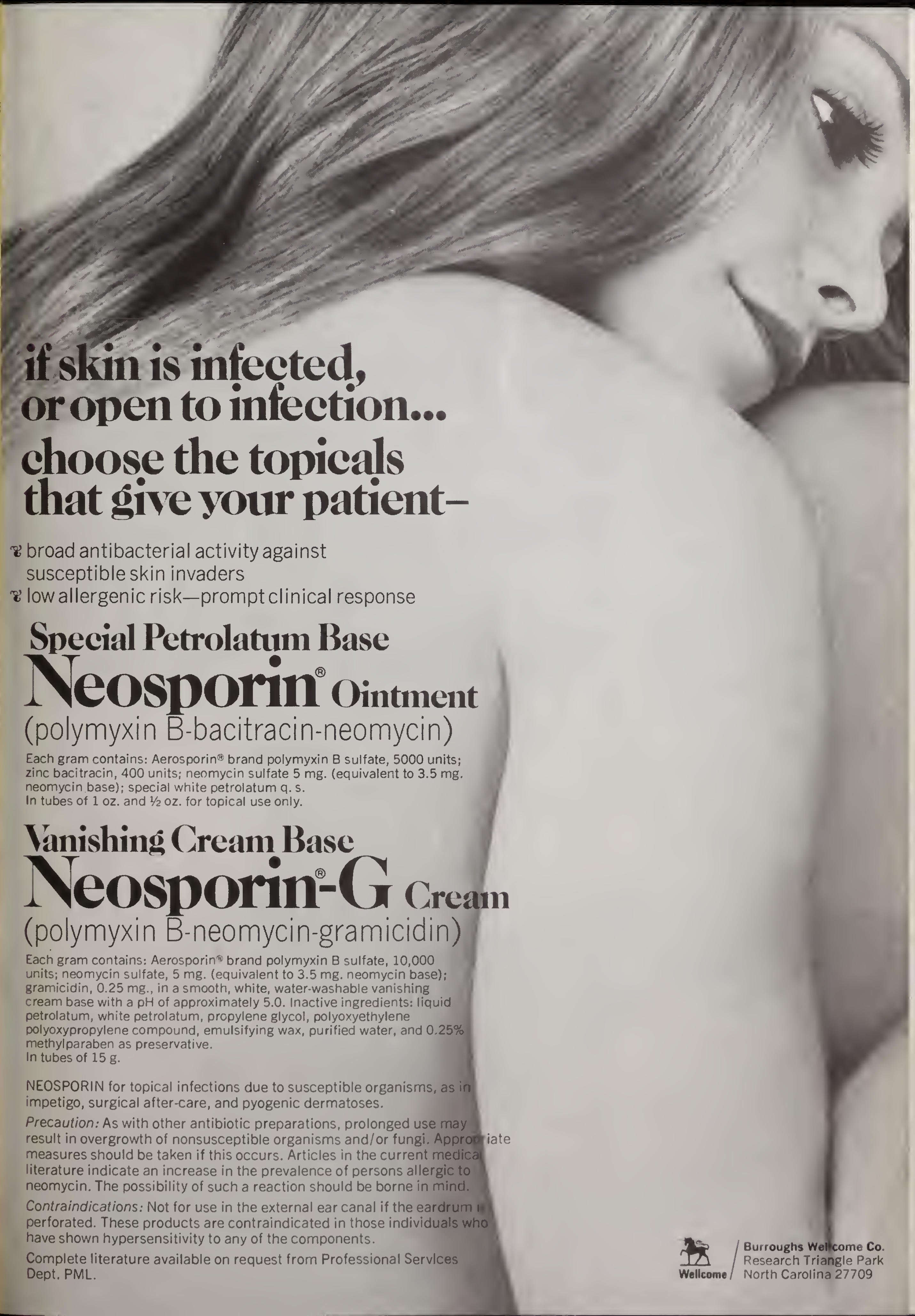
But the most important participation will be by the private practitioner. First, he will be asked to distribute appropriate literature to his patients. Second, he will be asked to check pelvic cultures on all female patients within appropriate age groups having pelvic examinations. Culture facilities will be provided through the Community Health and Hospital Administration. Third, he will be asked to report cases (by number, not specific identification). Fourth, he will be asked to pursue an energetic follow-up policy of contacts. Fifth, he will be asked to participate in and contribute to hospital staff conferences on venereal disease. Finally, he will be asked to contribute a few hours one afternoon or evening during the week to help man the extra clinic facilities in participating hospitals. Other forms of involvement may be suggested by the Committee as plans evolve.

Some forty years ago the Surgeon General of the Public Health Service, Thomas Parran, wrote a book entitled "Ten Million Americans Have It." The subject was syphilis - and in the two decades following its publication the venereal diseases almost disappeared. But now they are back - and Dr. Parran's closing admonition is again appropriate: "The problem is simple. Find and treat. The solution is not so simple but is quite possible if we all work like the devil to beat the Devil's own plague."

Love Needs Care

Darrell C. Crain



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Recent Advances in the Management of Chronic Renal Disease*

MAJOR WILLIAM A. BRIGGS, MC, USA, AND MAJOR JAMES H. KNEPSHIELD, MC, USA

Not so long ago a patient presenting evidence of chronic renal failure had an inevitably dim prognosis. The patient's physician, likewise, had nothing to which to look forward in being able to offer the patient more than sympathetic support. Thus, there was little gratification from the management of this problem, and the patient was miserable during the last months of illness. Today this situation has changed, largely as a result of more successful long-term dialytic treatment and the success rates achieved by transplantation in recent years. Now there is the prospect that the patient with end-stage renal disease need not die, but instead may be rehabilitated to return to work, support the family, raise the children, and even enjoy variable degrees of recreation. Now the physician has new options available to him in the management of the patient and has reason to make every effort to insure optimal results if dialysis and transplantation are undertaken. It is the purpose of this presentation to discuss selected developments which should influence the primary physician's early as well as late management of patients with progressive renal failure. Although selected references will be provided in the bibliography, we regret that space will not allow citing the works of many deserving contributors to our fund of knowledge.

A universal feature of chronic renal insufficiency is anemia of variable severity. In the absence of complicating iron or folate deficiencies, the anemia is of the normochromic, normocytic variety and, although associated with shortened red blood cell survival, it is mainly the result of impaired erythropoiesis.[1,2] Ordinarily, the anemia develops gradually as renal mass decreases; thus, the patient is able to adapt to the decreasing hematocrit and to continue working and participating in other activities. It is important for several reasons, for the physician to permit this adaptation and to avoid the temptation to treat mild symptoms or the numerical hematocrit value with transfusions. First, the effect of the transfusion in raising the hematocrit is short-lived, and the hematocrit soon returns to the basal level; and there is evidence that transfusions may further depress bone-marrow activity in uremic patients,[3] thereby creating a need for subsequent transfusions. Second, there is the risk of transmitting hepatitis virus. This will probably become less of a problem with more widespread screening of donor blood for hepatitis-associated-antigen,[4] or with a greater availability and utilization of frozen blood, the use of which has been associated with a decreased incidence of post-transfusion hepatitis.[5] Third, a problem unique to the administration of multiple transfusions to chronic renal failure patients is sensitization to transplantation antigens which may result from repeated exposure to the surface antigens of platelets and leukocytes infused with erythrocytes. It has been shown that such pre-sensitization seriously jeopardizes the chances of successful transplantation, especially from cadaveric donors,[6] leaving the patient with no alternative to chronic hemodialysis for the rest of his life. Since leukocyte-poor, packed erythrocyte preparations decrease the mass of antigenic exposure, potential transplant recipients should be transfused with such preparations preferentially unless whole blood replacement is necessary.

Pre-sensitization may occur, however, even with leukocyte-poor transfusions, and it remains to be seen whether the use of frozen blood, which may be entirely free of platelets and leukocytes, will provide the ultimate solution to this problem. Although the use of frozen blood is expensive, its advantages in preventing sensitization and therefore subsequent transplant failure and repeated or prolonged hospitalizations could readily justify the additional expense.

The above points are not presented to suggest that transfusions should never be administered. The patient with coronary artery disease will develop increasing anginal episodes when the hematocrit decreases beyond a point and will therefore require occasional transfusions. Some patients develop progressive congestive heart failure with more severe degrees of anemia, and there are other patients who become unable to continue jobs requiring physical effort when their hematocrit falls; such patients should also be supported. It is important to

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be aware of the above objections, however, with regard to routine transfusion for minimal or vague symptoms or just because the hematocrit falls to a certain numerical value. The expected benefit from each transfusion should be carefully considered in each case. In addition, when transfusion is indicated, one should recognize the need to select blood products carrying the least risk to the patients and their future management.

As experience with maintenance hemodialysis increased, it became apparent that an increasing proportion of patients would develop bone disease as time passed,[7] even though they had no clinical evidence of skeletal involvement when dialysis was initiated. This discouraging failure of dialytic therapy stimulated more research into disordered calcium physiology in uremia, and considerable progress has been made in our understanding of this complication.

Progressive renal insufficiency is associated with 2 well-defined changes in function which affect plasma calcium concentration. With decreasing glomerular filtration there is a progressive tendency to accumulate phosphate ions; the latter combine with calcium ions and cause a decrease in ionized calcium concentration. In addition, impaired calcium absorption from the gut has been demonstrated in patients with renal failure due to impaired intestinal activity of vitamin D.[8,9] It has recently been demonstrated that the kidney is the major source of an enzyme which converts the vitamin D metabolite 25-hydroxycholecalciferol to 1,25-dihydroxycholecalciferol,[10] the metabolite considered to be primarily effective in enhancing intestinal absorption of calcium.[11] With decreasing renal mass, patients with chronic renal disease presumably produce progressively less 1,25-dihydroxycholecalciferol and become unable to absorb adequate amounts of dietary calcium. This also creates a situation in which normal ionized calcium concentration may be difficult to maintain without drawing on skeletal reserves. Decreased ionized calcium concentration from either phosphate retention or decreased calcium or both results in chronic stimulation of the parathyroid glands in these patients in order to both mobilize calcium from bone and to decrease the renal tubular reabsorption of phosphate. The latter effect allows those nephrons which are functioning to excrete a larger fraction of their filtered phosphate load.

The histologic skeletal abnormalities found in patients with renal failure include both osteomalacia and osteoporosis,[12] one being more predominant in some patients than others or at different times in the course of individual patients. The pathogenesis of these abnormalities can readily be appreciated from the disordered vitamin D metabolism and secondary hyperparathyroidism alluded to above. Potential approaches to therapy or to preventive management also become apparent. Even at early stages of renal insufficiency when most dietary restrictions are not necessary, it may be advantageous to begin progressive restriction of dietary phosphate intake to balance the patient's ability to excrete phosphates. Slatopolsky et al[13] have shown in dogs with chronic renal insufficiency that such an approach ablated the progressive increase in plasma parathormone level seen in animals given an unrestricted phosphate intake. In patients with more advanced renal insufficiency in whom serum phosphate is abnormally elevated despite dietary restrictions, it has become very clear that phosphate absorption must be decreased by prescribing phosphate-binding antacids with meals in doses sufficient to maintain a normal serum phosphate concentration. If such measures are not undertaken, the severity of secondary hyperparathyroidism progresses[14] and patients develop symptomatic bone disease and metastatic calcifications in a variety of organs and tissues.[12] They may also be at greater risk of developing skeletal complications after transplantation.[15]

Patients who have moderately advanced renal failure and decreased renal mass may also be candidates for supplementary calcium intake and possibly even vitamin D therapy with proper precautions. Patients most likely to benefit from this approach are those with clinical or biochemical evidence of a predominantly osteomalacic skeletal lesion. However, other patients with a low serum calcium may be given additional calcium as long as serum phosphate concentration is controlled by diet or antacid therapy or both as necessary. With regard to therapy with vitamin D, caution is urged. Some authors have been satisfied that such therapy is beneficial;[16] again, however, serum phosphate must be controlled and the patient must be followed carefully for the development of hypercalcemia and metastatic calcification. When the specific metabolite, 1,25-dihydroxycholecalciferol, has been studied further and becomes generally available, it may provide a more satisfactory, selective agent for the treatment of impaired calcium absorption.

It is not the intent of this discussion to review well-established concepts in the management of chronic renal failure.[17-19] However, it is an opportunity to re-emphasize their importance in terms of the total support of the patient in the hope that satisfactory rehabilitation can be maintained or obtained when the patient requires additional treatment with dialysis and transplantation, if possible. Adequate control of hypertension to prevent progressive vascular damage, avoidance of, or modification of the dose of potentially nephrotoxic agents, careful attention to dehydration as well as overhydration, and treatment of urinary-tract infection represent areas of particular importance which are beyond the scope of this paper. Considerable research is and has been in progress to improve instrumentation, techniques, and therapies in order to make long-term maintenance hemodialysis and transplantation successful and acceptable therapeutic modalities open to renal patients and their primary physicians. It is worthwhile to refer these patients for evaluation for dialysis, and possibly future transplantation, as soon as adequate conservative management fails to maintain them in a reasonably healthy and asymptomatic state. Good long-term results are likely to be achieved under these circumstances rather than when the patient has suffered the ravages of poorly controlled, chronic uremia.

Conclusion

The patient with chronic renal disease now has a reasonable chance for a qualitatively satisfactory increased survival because of increasing successful utilization of maintenance hemodialysis and transplantation. Recent information from a variety of disciplines suggests ways in which the patient's primary physician can provide more comprehensive management early in the course of illness which may profoundly influence the chances of success when more sophisticated supportive measures are required.

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The Nitroblue Tetrazolium Dye Test*

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Reduction of nitroblue tetrazolium dye by polymorphonuclear cells was found originally to be a useful test for diagnosing and studying patients with chronic granulomatous disease.[1,2] Later it was adapted to the study of patients with a variety of febrile illnesses. Preliminary reports showed it to be very promising in distinguishing febrile patients with acute bacterial and fungal infections from those patients with fever from other causes.[3-6] The following is a report of our experiences with the NBT dye test over a 6-month period.

Method

The nitroblue tetrazolium dye test was modified from the method of Park.[3] Five ml of the patient's blood was mixed with 200 USP units of sodium heparin (Upjohn Co., Kalamazoo, Mich.). Plastic syringes and plastic test tubes were used throughout.

An equal volume of heparinized blood and nitroblue tetrazolium dye were incubated for 30 min. at 37°C. A thin smear was then stained with Wright's stain and the number of neutrophils containing reduced black formazan deposits was determined after viewing 100 neutrophils. All neutrophils with distinct deposits were considered positive.

The entire test was performed within 90 minutes. Preliminary studies showed that there was no deterioration of neutrophil stability within 90 min. of venipuncture. Prior to each use the dye was filtered through a 0.45 μ millipore filter (Millipore Corp., Bedford, Mass.) as recommended by Chretien.[7] The WBC and differential were performed on each blood sample by routine methods.

A stimulated control with 200 μgm/ml endotoxin lipopolysaccharide B *E. coli* 026:B6 (Difco Laboratories, Detroit, Mich.) as described by Park was included in all but 20 of the tests performed.

Patients were chosen for study if they had fever, leukocytosis, or both. Only those patients in whom the etiology of the fever could be determined by laboratory studies or clinical means (i.e., Hodgkins disease, Guillain-Barre syndrome) were included in the study.

Patients were classified into 3 groups based on a modification of Feigen's nomogram.[4,10] The formula for determining the total number of NBT positive PMN's is as follows: total WBC x (% PMN's + band forms) x % positive PMN's. Patients falling into the "A range" were considered normal, those in the "B range" were considered partially treated for bacterial or uncomplicated viral infections, and those in the "C range" were considered to have an active untreated infection.

Salicylate blood levels were determined by the methods of Trinder.[8]

Results

A total of 117 patients were studied and were categorized clinically as shown in table 1. Any patient who had received antibiotics prior to the NBT test was considered "partially treated". The great majority of these patients were studied within 24 hours of the institution of therapy. No patients were classified as "cured", since this must be a subjective judgment in many cases.

Those patients who had clinical evidence of infection for more than 3 weeks were considered to have a subacute or chronic infection.

Eight of the 12 patients who had acute untreated bacterial infections fell into the "C range".

Two of the 3 patients who fell into the "A range" had bacteremia. The first patient was receiving heparin for thrombophlebitis by continuous intravenous infusion. Because of a temperature elevation to 104°F (40°C), blood cultures were obtained and his IV discontinued. Twelve hours later he became afebrile. It was at this time that the NBT dye test was obtained.

The second case was a postpartum woman who had positive blood cultures for Bacteroides species on 2 separate occasions but had become afebrile and clinically well without antibiotic therapy. Her NBT dye test was normal at the time of her second positive blood culture. Subsequent blood cultures were sterile. She has remained asymptomatic for more than 3 months.

The third case was admitted because of high fever and petechiae. Clinical and laboratory examinations, including an LP, were normal except for the isolation of a group-A beta hemolytic streptococcus on a throat culture.

An additional case fell into the "B range". A blood culture was positive for beta hemolytic streptococci, and an acute bacterial sinusitis was found in a renal-transplant patient on therapy with prednisone and imuran. However, he had normal NBT dye tests prior to and after the acute episode (table 2).

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Twenty-two patients were classified as "partially treated". Five of the 6 patients in the "A category" had been receiving appropriate antimicrobial therapy for 72 hours or more with good clinical response. A sixth patient died within 18 hours of developing a bilateral aspiration pneumonia and disseminated intravascular coagulation syndrome. One of the 13 other patients in the "B range" had a chronic loculated infection of the pericardium. The remaining 17 patients had been on appropriate therapy for 12 or more hours. Two of the 3 patients who fell into the "C range" had received appropriate treatment for less than 12 hours, while the third had a gram-negative (*E. coli*) pneumonia under appropriate therapy for 48 hours.

Five cases of chronic untreated infection were studied (chronic osteomyelitis, chronic urinary-tract infection, chronic liver abscess, and SBE, 2 patients). Three fell into the "A range" and 2 into the "B range".

Control patients consisted of 14 healthy adults (aged 21 to 69) and 13 institutionalized elderly patients (aged 55 to 96). Also included were 6 patients who were clinically well and afebrile but hospitalized for evaluation of fibrous dysplasia of bone, allergic rhinitis, Gilbert's syndrome and Hodgkin's disease (3 patients).

Eight of the 9 cases of viral infection were documented by viral isolation, antibody titer rise, or both. The ninth case had a history and liver biopsy compatible with type MS-1 hepatitis.

As expected, the patient with chronic granulomatous disease had a NBT dye test of 0% and his cells could not be stimulated to reduce the dye with endotoxin.

Fifteen patients with noninfectious fevers were studied (table 3). The 1 patient whose results were in the "C range" had taken an overdose of aspirin. His NBT remained elevated for over 48 hrs. despite recovery, but he could not be followed. Because of this experience, 8 laboratory volunteers ingested aspirin to study its effect on the NBT test. In table 4 it can be seen that, despite significant salicylate blood levels, none of the volunteers developed abnormal NBT dye tests.

Three patients were classified as having neutrophilia because of persist-

TABLE 1. Nomogram Distribution of Results in Patients Studied with NBT Dye Test.

	Classification			Percentage of positive neutrophils	
Condition with Pos. NBT	A	B	C	Range	Mean
1. Acute bacterial infection					
Untreated	3	1	8	3-74	30
Partially treated	3	13	3	2-40	18.4
2. Subacute + chronic Infected (untreated)	3	2	0	0-29	11.2
Condition with Neg. NBT					
1. Normal Controls	31	2	0	0-17	6.6
2. Uncomplicated viral infections	9	0	0	1-28	8.1
3. CGD	1	0	0	0	
4. Noninfectious fever	13	1	1	0-34	6.3
5. Neutrophilia without fever	4	0	0	0-1	–
6. SLE	2	0	0	3-15	
Condition of Uncertain Status					
1. Parasitic infections	3	0	1	2-38	
Hookworm	1			8	
Leishmaniasis (cutaneous)			1	38	
Amebic abscess (treated)	1			2	
Pneumocystis Carini	1			16	
2. Synphilis (tertiary)	1			2	
3. Tuberculosis	2	0	0	2-3	
4. Fungal (chronic cryptococcosis)	1	0	0	4	
5. Malaria (treated)	1	0	0	16	

TABLE 2. NBT Dye Test Before and After Infection.

Date	NBT Score	WBC mm^3	Poly's	Range	Comment
Jan 12	0%	19.1	88	A	
Jan 14	5%	23.3	94	B	Pos. β-strep blood cult.
Jan 17	24%	10.6	77	B	Sinus drained surgically; penicillin administered
Jan 21	0%	17.9	97	A	Well
Jan 24	0%	14.5	95	A	Well

TABLE 3. Noninfectious Fevers

Diagnosis	Range A	Range B	Range C
1. Acute myocardial infarction	2		
2. Hyperthyroidism	1		
3. SLE with renal involvement	1		
4. Goodpasture's syndrome	1		
5. Cirrhosis with alcoholic hepatitis		1	
6. Overdose of salicylates			1
7. Pulmonary embolism	1		
8. Neoplastic disorders			
Metastatic malignant melanoma	1		
Metastatic carcinoma of rectum	1		
Carcinoma of rectum	1		
Brain tumor	1		
Metastatic breast carcinoma	1		
Multiple myeloma	1		
Metastatic neuroblastoma	1		

TABLE 4. Effect of Salicylates on the NBT Dye Test.

Patient	% Mg Salicylate level	% NBT Score
Overdose	85	34
	61	49
	13	54
Volunteer		
1	19*	3
2	40	3
3	18	2
4	25	2
5	19	6
6	23	2
7	22	4
8	24	8

* Highest level of salicylate reached

ently elevated neutrophil counts (> 10,000/mm^3) for a period of 1 or more weeks. All of these patients had NBT scores within the "A range".

The test was also evaluated as a means of following the response to therapy in some patients. One of these cases has been presented above (table 2). Five other patients with acute untreated infection were also studied, and in each case their NBT dye tests returned to normal.

Discussion

Our results agree in general with the previous studies reported in the literature.[3-6,9] The NBT dye test appears to be helpful in alerting the physician to the possibility of an acute bacterial infection. In this sense, it appears to be more helpful than the white blood cell count and differential or the fever pattern. However, it has to be stressed again that the NBT dye test must be interpreted in each individual case in the light of the clinical examination and other laboratory findings.[5,6]

In considering the entire group of untreated bacterial infections, half of our cases fell into the "A" and "B" ranges. However, when we separated the patients into acute vs subacute and chronic infections it was the latter groups NBT results were entirely in the "A" or "B" range.

We have speculated that this is due to the development of a tolerance of the neutrophil to the infecting microorganism. In each case the patients' PMN's were stimulated well with endotoxin suggesting that no underlying defect of the PMN's was present. Also, 1 of these patients sequentially developed a significant NBT shortly after therapy was instituted.

With regard to those patients with acute untreated bacterial infections, there were 4 patients who were classified in the A or B ranges and thus represent NBT "failures". Two of the patients with an acute bacteremia were in the "A" range. However, in 1 the NBT dye test was done after the probable source of infection was removed and the patient had become afebrile. Matula[5] showed that the NBT dye test may return to normal before the clinical response has returned to baseline. Perhaps removal of the IV had already cured this patient prior to his NBT dye test. The second patient had a positive blood culture when he was clinically well and had a normal WBC and NBT dye test. She never received antibiotics during or after her hospitalization. Subsequent blood cultures were sterile, and she has remained well for at least 3 months. The clinical course suggests that the bacteremic episodes were transient and without significant residua.

The third patient may have had a viral infection and may have been a carrier of group A Beta-hemolytic streptococci. No viral studies were done.

As mentioned, the infected untreated patient who fell into the "B range" was on Prednisone therapy. Several authors[11-13] have remarked that steroids tend to "normalize" the NBT dye test. However, this patient had a previous NBT study performed in which there was a significant rise and then fall after therapy. In selected patients receiving steroids serial NBT determinations may be a useful way of monitoring for infection. None of the other patients in the infected groups were receiving steroids. However, 1 of the patients with a documented influenza viral infection was being treated with steroids. In preliminary studies with patients receiving steroids we have found that their WBC's could be stimulated with endotoxin to the same extent as normal patients.

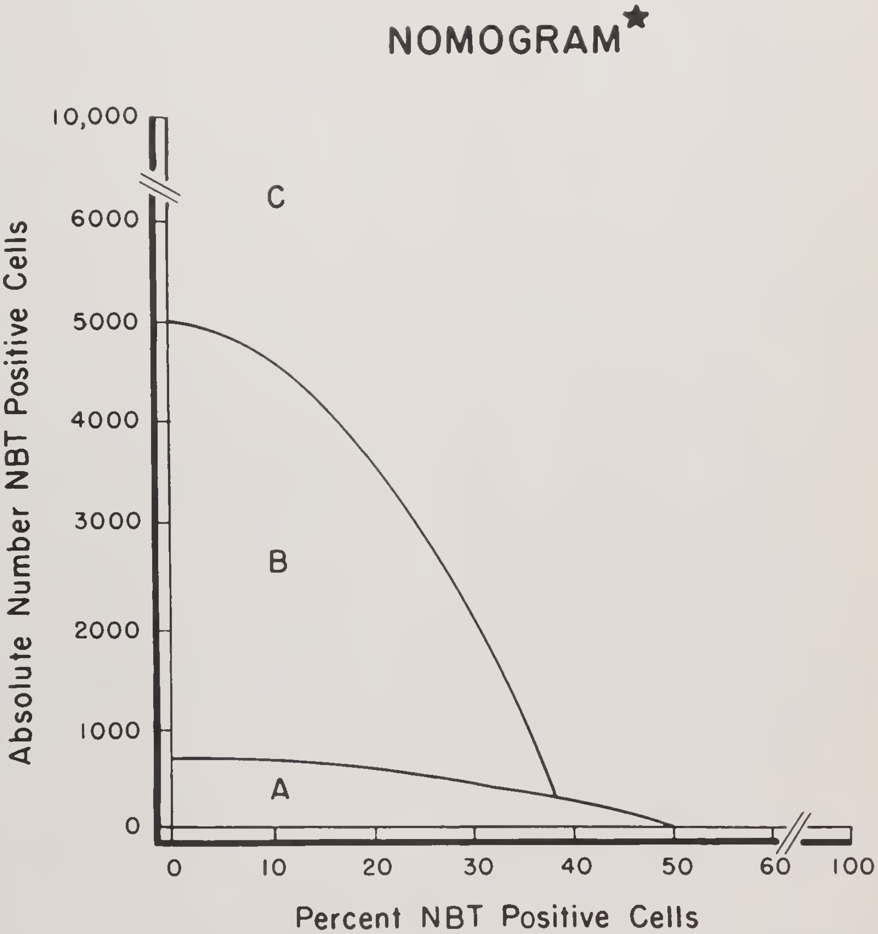
Our studies have corroborated Feigin's[4] and Matula's[5] observations that the NBT dye test is useful in monitoring a patient's response to therapy. In addition, 2 of the 3 patients classified as partially treated and which fell into the "C range" had received therapy for less than 12 hours, while those in the "B range" had been receiving appropriate therapy for more than 12 hours.

Five of the 6 patients who fell into the "A range" were clinically well. The sixth patient died in shock 18 hours after an episode of aspiration and was receiving large doses of steroids at the time the NBT test was done.

We also have found that the nomogram is extremely useful in interpreting the results. For example, 1 of our control patients had an NBT value of 17% with a WBC of 6,000, 26% of which were PMN's, while a patient with a documented varicella infection had an NBT score of 28% with a WBC of 2,900 and 34% PMN's. If these NBT scores were taken alone, both of these patients would have been considered positive. Using the normogram both results were in the A category.

Park[14] suggested that stressing the PMN's with endotoxin would be helpful in interpretation, particularly in ruling out underlying PMN defects. This technique may also help to shed some light in the underlying pathophysiology of some types of infections. Other investigators have studied this aspect and have found it useful.[5,12] We have now made this part of our routine NBT dye test.

Patients with malaria,[5,9,12] various parasitic[9] and fungal[3] infections have been found to be positive by other investigators. The situation with tuberculosis is unclear. Early reports suggested that these patients had normal NBT dye tests.[5] However, Mandell,[16] using a different method found patients with tuberculosis to have significantly higher NBT scores which in selected patients returned toward normal after treatment.



A - Normal subjects

B - Viral infection, partially treated bacterial infection, non-infectious febrile illness

C - Untreated bacterial diseases

★ Modified from Feigin

When we found such high NBT values in our patient with salicylism and recognizing that salicylates are commonly used as antipyretics, we investigated the effects of salicylates on the NBT dye test in normal volunteers. The results in table 4 indicate that there is no appreciable salicylate affect on the NBT test by the method we employed. The elevated NBT in the 1 individual may represent an idiosyncratic reaction or a unrecognized infection.

Our results have not been as rewarding as those previously reported.[3-5] However, our false negative cases all appeared to have plausible explanations for their low values. Also, the incidence of false positives was very low (<1%).

Dividing the patients into categories as we and others have done has obvious drawbacks when faced with the acutely ill patient. Nevertheless, it must be kept in mind that this test measures certain metabolic functions of leukocytes[1,2,17,18] and will be positive whenever these metabolic systems are stimulated. Since acute bacterial infection is a very common stimulus, the NBT dye test can be extremely useful as an adjunct to diagnosis and treatment of bacterial infection.

Conclusion

The nitroblue tetrazolium dye test is a helpful adjunct to the study of febrile illnesses. A value which falls into the "C range" most often signifies an acute bacterial infection, while values which fall into the "A or B ranges" are most often seen with other causes of elevated temperature. The test is also useful in following the clinical course of some patients.

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Current Concepts of Immediate (Reaginic) Hypersensitivity*

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We are indebted to von Perquit for recognizing the common denominator between immunity and hypersensitivity and for introducing the term "allergy.[1] In 1906 he wrote, "For this general concept of a changed reactivity I propose the term allergy". Hypersensitivity and immunity then are descriptive terms which are not mutually exclusive and which identify a clinical state of allergy.

The animal host is capable of mounting hypersensitivity responses which can be manifest immediately (within minutes), later (within hours), or delayed (within 2 to 3 days). In addition, the plasma constituents and cellular responses involved in these different manifestations of hypersensitivity are peculiar to the type of response.

It is the purpose of this review to discuss our current knowledge and understanding of the immediate (reaginic) hypersensitivity reaction. These reactions are characterized by a rapid release of short-lived pharmacologically active agents. These agents are released by tissue cells after a reaction between the antigen (foreign substance) and the antibody (immunoglobulin) which is attached to the cell surface. Anaphylaxis is an immediate hypersensitivity reaction. Allergic rhinitis and allergic asthma are other examples of immediate hypersensitivity reactions seen in man.

The complete mechanism of immediate hypersensitivity reactions is visualized as a series of reactions. These include: 1) fixation of an antibody to the cell surface, 2) binding of antigen to this antibody with resultant conformational changes in the antibody, 3) activation of an energy requiring enzyme system, and 4) a secretory response of mediator release (Fig. 1).

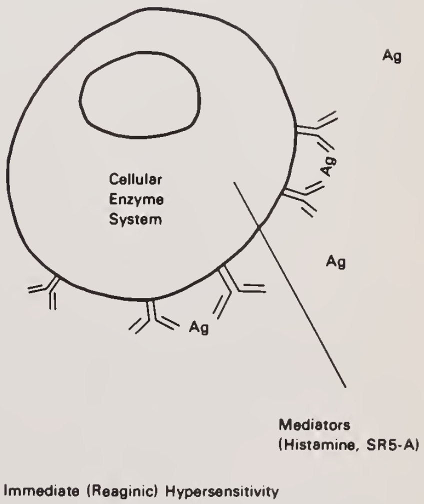

FIG. 1. Immediate (Reaginic) Hypersensitivity.

Immunoglobulin E and Reaginic Antibody

The antibody in immediate hypersensitivity reactions is reaginic antibody. It is found in the serum of allergic patients and is characterized by the ability to sensitize human skin. This was first demonstrated in the experiments of Prausnitz and Küstner in 1921.[2] The serum of fish-sensitive Küstner was injected intradermally into the skin of Dr. Prausnitz. The testing site was challenged 24 hours later with an extract of fish, and the response was an immediate wheal and flare reaction. This sensitizing activity by passive transfer of human reaginic antibody is generally limited to the tissues of man, ape and monkey. In addition to skin, passive sensitization with human reaginic serum of human leukocytes and human and monkey lung-fragments has been achieved (Levy and Osler, 1966;[3] Goodfriend, Kovacs and Rose, 1966).[4]

The presence of Immunoglobulin E (IgE) in human serum was first demonstrated in 1966 by Dr. Kimishige Ishizaka and his wife Dr. Teruko Ishizaka.[5] This was done in the laboratories of the Childrens Asthma Research Institute and Hospital, Denver, Colorado. Working with the serum of allergic patients, these physicians were able to identify IgE as the carrier of reaginic antibody activity.

Soon thereafter an IgE myeloma protein was first detected (Bennich and Johansson, 1967).[6] The availability of this protein in high concentrations enabled physicochemical analysis. IgE is a glycoprotein with an electrophoretic mobility of gamma I globulins. It has a sedimentation coefficient in the ultracentrifuge of 8.0 and a molecular weight of approximately

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200,000. As is found in most immunoglobulins, the IgE molecule is composed of 2 light chains of amino acids, and 2 heavy chains (Fig. 2). The antigenic determinants specific to the IgE Class of immunoglobulins are found in the F(c) portion of the molecule, and the antibody combining sites are in the F(ab) portion. The immunoglobulin binds to the cell surface by the F(c) portion (Ishizaka, K. et al 1967a, 1967b).[7,8]

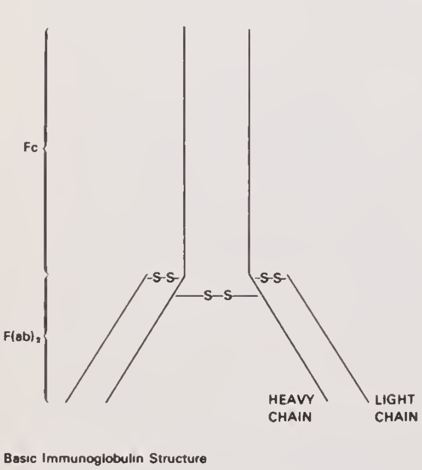

FIG. 2. Immunoglobulin Structure.

IgE-forming plasma cells are found in the respiratory and gastrointestinal mucosa, in tonsils and adenoids, and in bronchial and peritoneal lymph nodes (Tada and Ishizaka, 1969).[9] Small quantities of IgE have been found in nasal secretions of allergic patients (Tse et al, 1970).[10]

The most important immunochemical property of IgE is the ability to fix to target cells of immediate hypersensitivity. These target cells are the basophil in peripheral blood and the mast cell in tissue. When a monkey is given an intravenous injection of radiolabeled IgE myeloma protein (I^{125}-IgE) the label can subsequently be demonstrated attached to mast cells in the omentum and in the lamina propria of the small intestine and bronchi (Ishizaka, K., et al, 1971).[11] When human peripheral leukocytes are incubated with I^{125}-IgE the label is found attached to the basophils. If these basophil-fixed antibodies are challenged with antigen the basophil loses metachromatic granules and histamine is released (Ishizaka, T., et al 1971).[12]

Immunoglobulin E is present in very small amounts in normal human serum. The level slowly rises from a few nanograms per ml at birth to a few hundred nanograms per ml in adults (Johansson, 1968).[13] Patients with allergic rhinitis, extrinsic (allergic) asthma and atopic eczema have significantly raised levels of serum IgE. The IgE level is also increased by allergen stimulation. During a pollen season increases of IgE are found in patients with asthma and hay fever. There is no definite correlation between IgE level and severity of allergic state, however. Very high levels of IgE (mcg per ml) have been found in parasitic infestations including Ascaris, Toxocara, hook worm and Echinococcus (Johansson et al, 1970).[14] Some human serum samples contain little or no measureable IgE. The significance of this is unknown.

Release of Chemical Mediators

Peripheral leukocytes of allergic individuals will release histamine on exposure to specific antigen (Lichtenstein and Osler, 1964).[15] When the white blood cells are isolated and exposed in vitro to varying concentrations of antigen requirement for induction of portion to the amount of antigen used and the sensitivity of the patient. The antigen histamine is released in proleukocyte histamine release in vitro is quite small and in the range of 0.1 to 0.00001 mcg of antigen protein per ml. This phenomenon of antigen-induced leukocyte histamine release provides an in vitro model of the allergic reaction steps. Furthermore, when a symptom-index score of the clinical severity of allergic sensitivity of the patient to a given allergen is compiled, this symptom-index parallels the in vitro sensitivity of the patient's leukocytes for histamine release (Lichtenstein et al, 1966).[16] The more severely affected patients require much less antigen in vitro to induce leukocyte histamine release.

In figure 3 it can be seen that the patient VnD required less ragweed antigen than patients GM and CH to elicit release of 50% of the leukocyte histamine. Patient VnD is also more sensitive to the airborne pollen by history and symptom score.

Sada et al (1969)[17] have demonstrated that immunotherapy with allergen extract results in a diminution of sensitivity of the leukocytes to the antigen as manifest by in vitro histamine release. These patients also have an improvement in the symptom-index score (Lowell and Franklin, 1965).[18]

Histamine is found in largest quantities in circulating basophils and tissue mast cells. When sensitized monkey or human lung fragments are challenged in vitro with specific antigen, histamine is released (Goodfriend et al, 1966). The sensitized lung fragments are obtained by using lung tissue of an allergic patient (Brocklehurst, 1960),[19] by incubation of normal lung fragments in reaginic antibody containing serum (Parish, 1967),[20] or by incubation of the fragments in E-myeloma serum. The antigen is one to which the patient is sensitive. An antisera prepared against IgE can also be used (Ishizaka, K. and Ishizaka, T., 1969).[21]

In addition to histamine, sensitized monkey and human lung fragments have been found to release slow-reacting substance of anaphylaxis (SRS-A) (Brocklehurst, 1960,[19] Ishizaka, T. et al,[22] 1969) and a factor chemactactic for eosinophils (ECF-A) (Kay and Austin, 1971).[23] SRS-A is an acidic lipid with a molecular weight of 900-1,250. It is identified by its ability to cause a slow, prolonged contraction of guinea-pig ileum in a Schultz-Dale

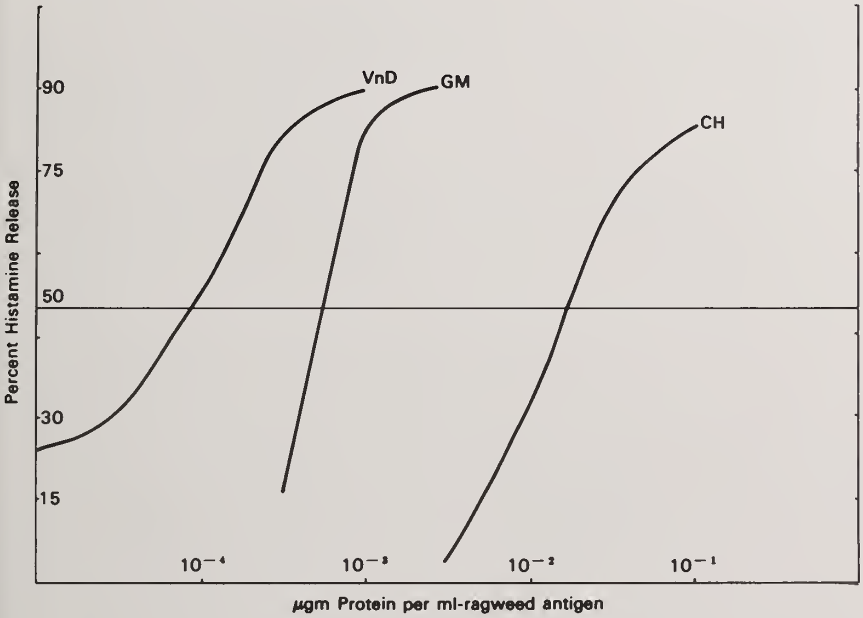

FIG. 3. Antigen induced leukocyte histamine release.

preparation. SRS-A is released from the lungs of asthmatics on challenge by specific pollen allergen, and it is a potent stimulator of human bronchial smooth-muscle contractions.

ECF-A was first described in 1971 (Kay et al).[24] This substance has a molecular weight between 500 and 1,000 and is also released from the lungs of asthmatic patients when challenged by antigen. ECF-A is specific for the attraction of eosinophils.

Pharmacologic Control Mechanism

The immunologic release of the forenamed chemical mediators from target cells is an energy-dependent process, most probably enzyme mediated (Osler et al, 1968).[25] Thirty-six years ago Schild (1936)[26] observed that epinephine inhibited the immunologic release of histamine from guinea-pig lung. This effect of catecholamines is now known to be by activation of an enzyme, adenyl cyclase, on the surface of the cell. This in turn increases the cellular content of cylic 3′-5′ monophosphate (CAMP) (Sutherland and Rall, 1960.[27]

This system of CAMP is a cellular secretory system and was first described by Dr. E. W. Sutherland in 1958.[28] Dr. Sutherland recently won a Nobel Prize for his work on CAMP.

Drugs capable of increasing cellular levels of CAMP (isoproterenol, epinephrine, methylxanthines, and corticosteroids) will inhibit the release of histamine and SRS-A (Lichtenstein and Margolis, 1968;[29] Keopman et al, 1970).[30] These drugs are therefore capable of directly modulating the IgE mediated immediate hypersensitivity response.

Our understanding of immediate hypersensitivity reactions has been enhanced tremendously in recent years. The knowledge gained and concepts developing offer new directions in our goal of relieving patients suffering from allergic disorders.

Summary

Immediate hypersensitivity reactions are characterized by a rapid release of short-lived pharmacologically active agents. This phenomenon is mediated by reaginic antibodies of the immunogobulin E class of immunoglobulins. The agents released include histamine from peripheral basophils and histamine and slow-reacting substance of anaphylaxis from tissue mast cells. A new mediator, eosinophil chemotactic factor, released by sensitized lung tissue has also described. The release of these mediators is dependent upon cell fixation of the antibody, binding of antigen by the antibody, and activation of cellular enzyme and cellular secretory mechanisms subsequent to the binding of antigen. Pharmacologic inhibition of this reaction sequence has been described.

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In-Vitro Methods of Detecting Drug Hypersensitivity*

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The study of adverse drug reactions is often a very difficult and confusing subject. Adverse reactions may be classified into various types depending upon the mechanisms involved.[1]

1. *Overdosage.* The untoward effects are a consequence of too high concentration of the drug in body tissues leading to over intensification of known pharmacologic actions and side-effects.
2. *Intolerance.* The reaction here is a qualitatively normal pharmacological effect which is quantitatively increased.
3. *Idiosyncrasy.* Here the observed reaction is qualitatively abnormal and is not the expected pharmacologic response. No immunologic mechanism is involved. An example would be bizzare and hyperactive behavior after taking barbiturates.
4. *Side-effects.* This term means undesirable but unavoidable pharmacological actions of drugs which are not the actions for which the drug is being used (i.e., drowsiness associated with antihistamines).
5. *Secondary effects.* This term represents indirect consequences of the pharmacologic action of the drug. An example might be activation of dormant tuberculosis after use of corticosteroids.
6. *Allergic or hypersensitivity reactions.* These reactions are the result of injurious immune responses induced by the drug or its metabolites through the production of antibodies or of sensitized lymphocytes.

Drugs elicit antibodies by becoming haptens. They usually do not have a large enough structure to be recognized as antigens by the immune system. Drugs or their metabolites bind co-valently with proteins in sera and then may be recognized as being antigenic, leading to production of sensitized lymphocytes and antibodies which may then cause the hypersensitivity response (fig. 1).

In order to demonstrate a true hypersensitivity response, therefore, one must be able to directly or indirectly demonstrate antibodies or sensitized lymphocytes to the given drug or its metabolites. This is often a very difficult task. Frequently the drug itself is not the antigenic stimulus, but a metabolite (s) is. Also,

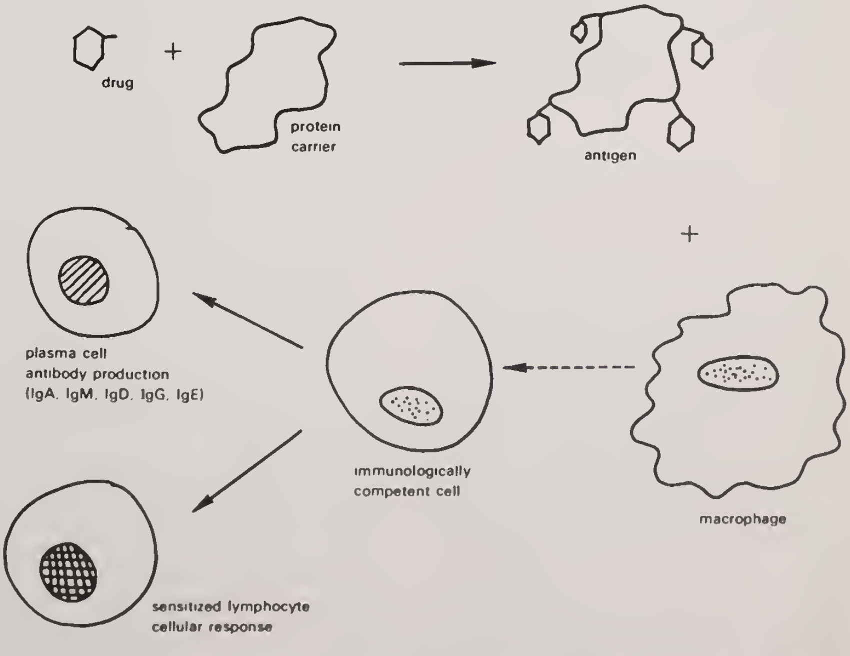

FIG. 1. Antibody production. The drug or a metabolite must bind with a protein to become antigenic. This protein-drug complex is then recognized as an antigen by the immune mechanism, and antibodies and sensitized lymphocytes are produced.

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antibodies produced against a drug hapten are not detectable in the serum indefinitely. In studies with penicillin allergy it has been shown that IgE against penicillin haptens is much easier to detect if the drug has been taken within 2 years of the time of testing.[2,3,4] The lack of antibody in sera when the patient has not been exposed to a drug antigen for a relatively long time does not mean that the patient will not develop an allergic response with re-exposure to that drug. In addition, the presence of antibodies (IgG and IgM) in sera of the patient does not necessarily mean allergy or hypersensitivity. The presence of IgE directed against the drug in sera, however, corresponds well with the presence of hypersensitivity. It has been shown with hemagglutination techniques, radioimmunoelectrophoreses, and complement-fixation tests that antibodies IgG and IgM are often demonstrated in sera of patients previously exposed to drugs who have had no demonstrable hypersensitivity reactions.[5]

The importance of adequate in vitro methods for demonstration of drug hypersensitivity has been stressed by many authors.[6,7,8] The commonly used in vivo techniques of skin testing, P-K transfer, and provocation testing have a number of problems.[1,5] Skin-testing may sensitize or actually induce allergy in patients, can precipitate a systemic anaphylactic reaction, and does not take into account the possibility that a drug metabolite is the offender instead of the drug itself.[1] P-K transfer is cumbersome, time-consuming, and carries the possibility of transmission of disease such as hepatitis. Provocation-testing is obviously time-consuming and possibly dangerous to perform. For these and other reasons the search for in vitro methods of demonstrating drug hypersensitivity has been intensifying recently. This paper discusses methods recently introduced and some now being evaluated for usefulness in in vitro diagnosis.

Basophil Degranulation Test

Investigators have recognized for quite a few years that tissue mast cells and basophil leukocytes undergo morphologic changes and degranulation during the acute anaphylactic type of allergic response (urticaria, asthma, anaphylaxis)[9,10] This observation and the accepted theory that antigen and tissue fixed IgE antibody reactions cause release of histamine and other mediators into surrounding fluid with the release of the granules from the mast cells and basophils led Shelley to choose these basophils as possible indicators for allergic reactions (Fig. 2).[11,12] The procedure involves incubating buffy coat of the patient's blood (direct method) or of rabbit blood (indirect method) with the patient's serum and an appropriate concentration of antigen (drug, pollen extract, food allergen, etc.). The mixture is then put on a slide and stained with toluidine blue, and 20-100 basophils are counted with the percentage of "altered" or degranulated basophils noted.

Some early reports with this procedure in drug allergy were very encouraging.[2,13] It soon became apparent, however, that this procedure had some pitfalls and limitations.[14,15] Investigators critical of the test note that there is no good correlation between the presence of clinical sensitivity or reaginic antibody and basophil degranulation.[15,16]

Even though this test may represent, as Shelley suggests, a miniature anaphylaxis model, the difficulties involved with getting good controls, lack of objective end-point, poor reproducibility, and poor association with reagin, make correlation of this test with atopic disease, including drug hypersensitivity, very difficult.

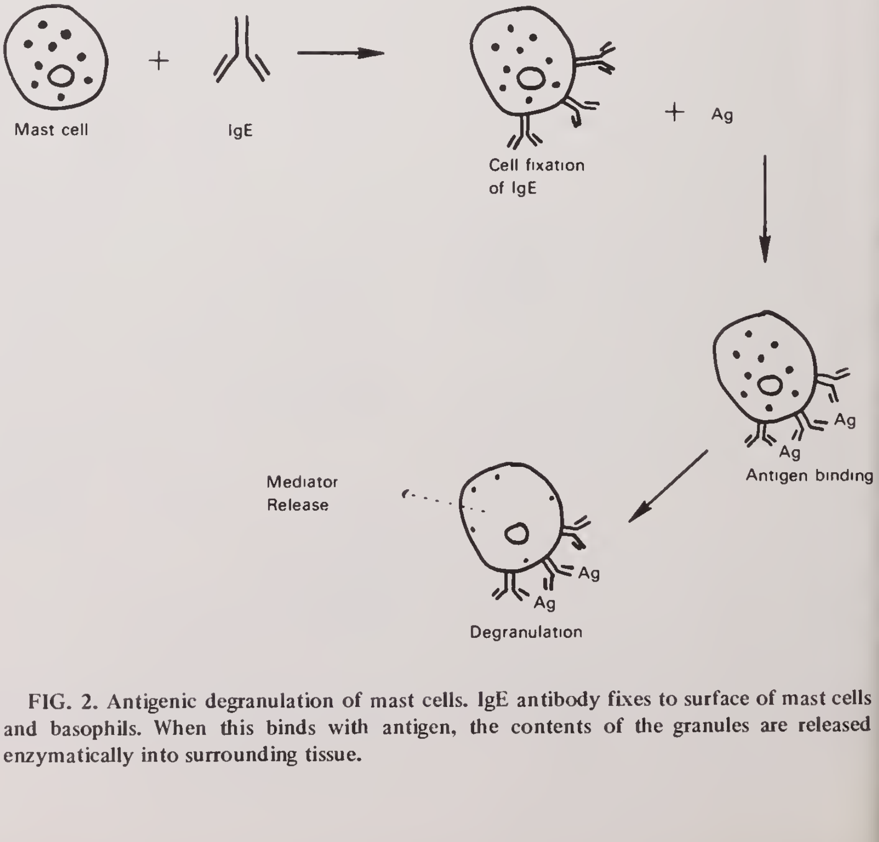

FIG. 2. Antigenic degranulation of mast cells. IgE antibody fixes to surface of mast cells and basophils. When this binds with antigen, the contents of the granules are released enzymatically into surrounding tissue.

Rat Mast Cell Degranulation

Schwartz and her collaborators[17] noted that the technique of identifying allergens with the basophil degranulation procedure of Shelley was very difficult to use and get reproducible results. They thought that this was quite often due to spontaneous or non-specific degranulation of the basophils making it difficult to get good controls. This led to a search for a different indicator cell to be used in the same system, and the mast cells from peritoneal fluids of rats were selected. The procedure involves sacrificing adult albino rats and injecting about 10 cc of collecting fluid into the peritoneal cavity. After 3 minutes this fluid is removed and centrifuged yielding peritoneal cells which are 3-5% mast cells.[7] These cells are resuspended in physiologic media. The patient's serum is then incubated with the mast cells, after which antigen (drug) is added and a slide is made of the preparation and stained with neutral red. From 40-200 mast cells are then observed for enlargement of cell, contorted outline with loss of granular staining, palisading of granules at periphery, or vacuolization and repulsion of granules from the cells. Allergic patients' sera yield 18-25% altered cells more than controls, while nonallergic groups give about a 7% or less altered cell count. Slides may be read as positive, also, if the counts are more than twice the control slide.

Further studies with this technique have given strong evidence that human reagenic antibody or IgE will fix to rat mast cells and that the IgE portion of human sera is the fraction associated with degranulation of the mast cells.[7,18,19,20] These studies, if confirmed, would indicate that this technique is an indicator for specific IgE in human sera and gives a sound theoretical basis for the use of the test. Studies done to date with penicillin and aspirin hypersensitivity have shown good correlation between this technique, history, skin tests, P-K transfer, and the radioallergosorbent test in diagnosis.[4,21]

In an effort to obtain a more accurate and objective measurement of the end-point in this experiment a method has been introduced which measures the amount of histamine released in the above technique with the degranulation.[19,20]

The rat mast-cell degranulation test is relatively easy to perform and has been reproducible in studies to date. The test also seems to show specific serum IgE against a given antigen and shows promise of being a good in vitro test for drug and other types of immediate hypersensitivity.

Radioallergosorbent Test (RAST)

The radioallergosorbent test was recently developed in Sweden and used for the detection of serum reagenic or IgE antibodies against specific allergens.[22] The test gives a relative quantitation of the IgE present in an individual's serum which is directed against the specific allergen being tested. Since IgE has been proven to be the primary antibody involved in immediate hypersensitivity reactions, a positive result would indicate that the individual would theoretically be susceptible to immediate hypersensitivity reactions and its manifestations of urticaria, angioedema, asthma, and anaphylactic shock if exposed to the allergen.

The method involves attaching the allergen or drug to insoluble particles (cellulose, sephodex, or paper discs). The patient's serum is then added to the particle-drug complex and incubated, allowing the antibodies in the serum to combine with allergen on the particle. After washing, anti-IgE labeled with I^{125} is added and incubated. This allows the radioactive labeled anti-IgE to combine with the IgE antibody attached to the particle-allergen complex. Counting the radioactivity of the particles and comparing with a standard sera then gives a relative quantitation of the IgE antibody present in the sera against the allergen (Fig. 3).

Studies with this test have shown that it correlates well with P-K transfer, skin tests, history, and provocation tests to foods, animals, and pollen in allergy diagnosis.[23,24,25]

A recent report with this technique for patients with a history of penicillin allergy showed good correlation with skin tests and provocation tests.[8] The authors thought it could replace the more dangerous skin test in prediciting patients who may have an immediate type allergic reaction with penicillin. This test reportedly can be done in great volume of up to 700 tests per week per technician,[26] but results must wait about 48 hours from the start of test.

Studies of this method with drug allergies are just now being done and results are not yet available. It appears that it might compare favorably with the more cumbersome and dangerous in vivo methods of P-K transfer and skin testing, and, if so, it would be a valuable tool. Mention should be made that purified anti-IgE used in the test is expensive and difficult to obtain in quantity.

Histamine Release

Another method of measuring immediate hypersensitivity is the leukocyte histamine-release study. It has been demonstrated very well that this procedure can be utilized to measure immediate hypersensitivity with ragweed antigen and leukocytes from ragweed sensitive patients.[27,28] The histamine comes primarily from the circulating basophils and is released by the same immunologic mechanism as occurs with mast-cell histamine release.

The method involves isolating leukocytes from the peripheral blood of patients suspected of having the allergy. The cells are washed and sus-

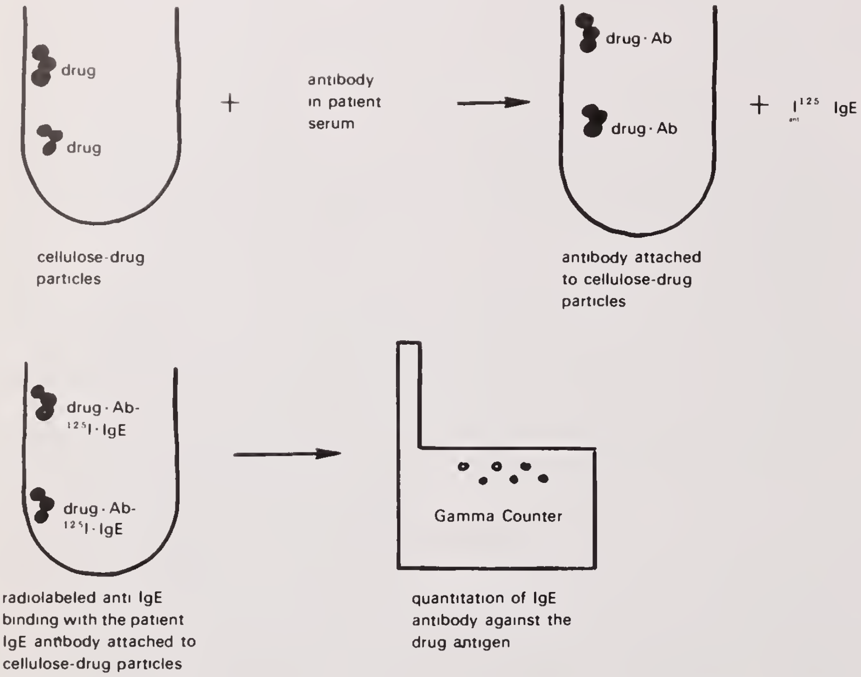
drug
drug
+
antibody in patient serum
drug · Ab
drug · Ab
+
I^{125} IgE
cellulose-drug particles
antibody attached to cellulose-drug particles
drug · Ab- ^{125}I · IgE
drug · Ab- ^{125}I · IgE
Gamma Counter
radiolabeled anti IgE binding with the patient IgE antibody attached to cellulose-drug particles
quantitation of IgE antibody against the drug antigen

FIG. 3. Rast test. The drug is bound to insoluble particles. Serum with antibody is added and antibody fixes to the drug-particle complexes. Adding anti-IgE labeled with radioactive iodine will cause the IgE fixed to drug-particle complexes to take up radioactivity.

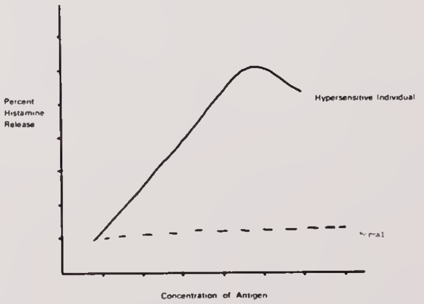

FIG. 4. Histamine release curves. This figure shows the amount of histamine released from leukocytes of an allergic individual after exposure of leukocytes to various amounts of antigen. A normal control is included for comparison.

pended in buffer and then incubated with antigen. The amount of histamine released into the supernatant is then determined by a fluorometric method and expressed as percent of total cellular histamine.[28,29] Results in atopic individuals are usually then reported as cell-sensitivity (concentration of antigen required to get 50 % histamine release and cell reactivity (concentration of antigen needed to get maximal release of histamine). Controls, or patients without hypersensitivity, to the antigen, will not release histamine (Fig. 4).

Leukocytes of 14 patients with anaphylactic or accelerated reactions to penicillin were tested by this method with penicillin G (Pen G) and penicilloyl polylysine (PPL) as antigens. Eleven of the patients responded to PPL or Pen G or both, and this correlated well with the skin tests.[30]

There has been very little work with this procedure in drug allergy, but it appears to have possibilities in diagnosing the immediate and, therefore, most dangerous type of reaction to drugs. Further work has to be done before final evaluations can be made. This technique tends to be long and tedious and would not be easily adapted to routine hospital use.

Lymphocyte Transformation (L.T.T.)

It is well known that human lymphocytes undergo transformation into blast cells when incubated in vitro with phytohemagglutinin or specific antigens to which the patient has developed previous immunity.[31,32] The application of lymphocyte cultures for immunologic studies has lead a number of researchers to the investigation of lymphocyte response to drugs in patients with known or suspected drug allergy.

This method involves separating the lymphocytes from peripheral blood. These cells are then incubated with a given concentration of drug for 4-6 days. At this time blast formation can be interpreted by microscopic examination of the lymphocytes or by "pulsing" with ^{3}H-Thymidine. The ^{3}H-Thymidine is incorporated into the nucleus of the cells at faster rates with increased blast formation and can be counted with a scintillation counter to give a good objective measurement of the degree of blastogenesis.[1]

The reports of the investigation into this area have been very conflicting. Halpern[33] studied 95 patients with a history of drug allergy and found positive L.T.T. in 86 of these with the microscopic method of counting. Patients with either immediate or delayed hypersensitivity responses were positive. Kunz[34] reported positive L.T.T. in 8 of 9 penicillen-allergic patients. Reichenberger and Heitmann[45] had positive responses in all 12 patients with tetracycline and ampicillen allergy histories. Other reports have been less convincing, with a much smaller percentage of patients showing positive L.T.T.[36,37] Still others report no correlation between drug allergies and L.T.T.[38,39]

There are some possible explanations for the differences seen in these reports. Most of the favorable reports were done by culturing relatively higher doses of drug with the lymphocytes and counting the blast formation microscopically. The more unfavorable reports were done by culturing with lower concentrations of drug and blast formation calculated with the ^{3}H-Thymidine method. All observers agree that the concentration of drug is important in the test procedure but that the values used are frequently different. ^{3}H-Thymidine incorporation is a much more objective determination of the L.T.T. than the microscopic method.

It would appear that the issue is still unsettled, but the more objective evidence seems to indicate a poor relationship between the test and drug allergy.

Macrophage Migration Inhibitory Factor (MIF)

Macrophage migration inhibitory factor (MIF) is a soluble mediator of delayed or cellular hypersensitivity. This factor appears to be a glycoprotein with a molecular weight of about 25,000-30,000 which can be assayed in culture supernatant after sensitized lymphocytes are cultured with antigen.[40] This assay is frequently used for in vitro detection of delayed hypersensitivity.[39]

The method consists of separating lymphocytes from the peripheral blood and incubating with appropriate concentration of allergen or drug. Each day the supernatant around the cells is changed and saved until 3 days have elapsed. The supernatant is dialyzed, lyophilized, and reconstituted. It is then assayed for MIF by measuring its ability to inhibit migration of guinea-pig macrophages from a capillary tube.[40]

Rocklin and David studied 11 patients with drug allergy involving 3 different drugs.[39] They found MIF was produced in 6 of the 11 patients. In 3 of these 6, lymphocyte transformation was done and was negative. In a related study Nordqvist and Rorsman[41] showed inhibition of leukocyte migration in 7 patients with drug-contact dermatitis using a different method.

More studies should be done in this area to get a better idea of the correlation between MIF production and drug allergy in a large number of patients. As suggested in one study, the technique may turn out to be useful in detection of drug allergy, but it seems unlikely that it could be commonly used for this purpose. There are occasional false negatives in MIF assays, and the procedure is expensive, long, and difficult to perform.

In summary, one could say that we are still looking for an easy accurate, and reproducible in vitro method of detecting drug hypersensitivity. The RAST test and rat mast-cell degranulation tests show possibilities for answering the need. It will be interesting to see how the controversy concerning L.T.T. and drug allergy will eventually be resolved and also to see results of further studies relating leukocyte histamine release to drug allergy.

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Peritoneoscopy: Has This Procedure Come of Age?*

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Historical Introduction

In 1901 when Ott[1] introduced a speculum through a culposcopic opening and viewed the abdominal cavity, using a head mirror as a light source, he heralded the birth of a diagnostic procedure which was destined to capture the imaginations of physicians on one side of the Atlantic and remain more or less dormant on the other side. He termed the procedure "ventroscopy."

A few months later at the German Biological and Medical Society's 73rd Session Dr. George Kelling[2] demonstrated a procedure on a dog which he called "coelioscopy." He used an optical instrument (a cystoscope) and a pneumoperitoneum needle. He subsequently performed the procedure on 2 human patients and has come to be considered the "Father" of peritoneoscopy.

Jacobaeus,[3] unaware of Kelling's work, re-described the procedure in 1910 and termed it "laparoscopy." He apparently performed the procedure on patients before Kelling and stressed its harmlessness when applied to patients with ascites.

In 1911, at Johns Hopkins, Bernheim[4] described his technique as organoscopy, using a proctoscope without the aid of pneumoperitoneum to visualize the abdominal cavity.

Over the next 1-1/2 decades scattered reports appeared in the literature from Europe and Argentina.[5,6,7] In 1920 Orndoff[8] published the first large series from the United States and popularized the term peritoneoscopy.

During the 1920's and 1930's Kalk,[9] Fervers,[10] and Ruddock[11,12] in this country introduced major innovations in the instruments and optics. Ruddock's instrument contained a separate biopsy apparatus built in the axis of the instrument.

Additional uses for peritoneoscopy began to be described. Its use in gynecologic diagnosis was mentioned by Korbsch in 1921,[13] and in 1937 Hope[14] reported the value of diagnosing ectopic pregnancy by this procedure. Articles by Cohen[15] and Fear[16] in 1968 and later by Siegler[17] and Harris[18] established the superiority of "laparoscopy" for gynecologic diagnosis over other methods of examination (i.e., examination under anesthesia, culdocentesis and colpotomy). Horwitz,[19] recently reviewed the literature on this subject.

Instrumental intervention combined with peritoneoscopy was first described by Fervers in 1930. He divided abdominal adhesions by cautery and removed tissues from the abdominal cavity for biopsy. A method of guided needle liver biopsy was evolved by Kalk in the early 1940's, and cholecystocholangiography was first applied by Royer and Solare in 1947.[20] Splenoportography, under peritoneoscopic control, was first described by Henning[21] in 1950, and gynecologic instrumentation, including sterilization by tubal cautery, dye studies, removal of foreign bodies and uterine suspension procedures began to be mentioned as early as 1941[22-28]

The improvements in German optics during the 1950's were applied to the peritoneoscope with success, and the advances in light transmission over the past 2 decades have been outstanding. From reflected light, to a distal "hot light" the path has led to the present system of delivering "cold" light from an external source, through fiberoptic bundles. With the advent of the internal flash, detailed color photography has become conveniently available.

Paralleling these developments, the procedure flourished in Europe and floundered in the United States. In Wittman's[29] extensive bibliography in 1960 approximately two-thirds of the 600 references are from the European literature. Roughly tabulating the total number of procedures performed, the ratio of those performed in Europe to those performed in the United States is about 10:1. Without the efforts of literally a handful of physicians in this country the ratio might be 50:1. When Parker and Hitzelberger[30] surveyed internists and surgeons in 1966, only half said they had ever witnessed a peritoneoscopy and only one-fourth felt it to be useful.

For a procedure which has been used for over 50 years on thousands of patients, the answers to the important questions should be evident. Is peritoneoscopy useful and is it safe?

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Discussion

Addressing ourselves to the above questions, the experience with peritoneoscopy at Walter Reed General Hospital over the past 8 years was reviewed.[31] During this period the procedure was performed on 450 patients and the results were in keeping with reports in the literature.

Technique. The technique of peritoneoscopy has been well-described by Ruddock,[32] Palmer,[33] and Palmer and Boyce.[34] The patient should be slightly pre-medicated. Whether room air, CO_2, or nitrous oxide should be used for the pneumoperitoneum is unsettled. Horwitz[19] discussed this issue congently.

The limitations imposed on peritoneoscopy are anatomic. The endoscopist should be as familiar with the limitations imposed by nature (i.e., the location of the pancreas) as he is with those that prior illness or surgery might present (i.e., adhesions). Unhampered by adhesions or excessive omentum, one should readily identify the anterior and inferior aspects of both lobes of the liver, gallbladder, anterior wall, and greater curvature of the stomach, the inferior pole of the spleen, the anterior aspect of both hemidiaphragms, most of the parietal peritoneum, portions of the small bowel, and cecum (occasionally the appendix), variable amounts of the colon, the urinary bladder, and the female pelvic organs. The inguinal rings and their contents can also be readily identified.

Adhesions are the nemesis of the peritoneoscopist. In the Walter Reed Hospital experience all 11 of the "unsuccessful peritoneoscopies" (2%) were due to postoperative adhesions, preventing a satisfactory pneumoperitoneum and/or visualization of the desired organ. Adhesions can distort and fix anatomic structures, increasing the risk of visceral injury and impairing visibility. In order to minimize the risk, the endoscopist must carefully select the incision site away from scars of prior surgery. During the establishment of the pneumoperitoneum, the operator must observe for asymmetrical enlargement of the abdomen. Once inside, if a view of the desired organ is totally impaired, there is little the endoscopist can do but examine other visible organs and withdraw the instrument. Smith[35] has suggested that filmy avascular adhesions may be safely separated by the instrument to permit a more adequate examination.

The usual diagnostic peritoneoscopy should take no longer than one-half hour. With teaching, directed liver biopsy and photography, the procedure can take up to 1-1/2 hours.

Indications. The classic indications for performing peritoneoscopy are well established[32] and articles in the American literature indicate their continuing validity and suggest additional indications.[37-38] The indications used at Walter Reed are listed in table 1.

Recent work has suggested still further indications. DeVita and Bagley[39] noted that peritoneoscopy is as useful as laparotomy in the staging of Hodgkin's disease.

TABLE 1

Indications

I. Hepatobiliary problems
  a. LFT abnormalities
  b. Hepatosplenomegaly
  c. Scan abnormalities
  d. Ascites
  e. ? Extrahepatic obstruction
  f. G. B. abnormalities

II. Detection of metastatic carcinoma

III. ? Other abdominal pathology
  a. Abdominal pain
  b. Abdominal mass
  c. Pelvic disorder
  d. Peritoneal disease
  e. Splenic disease

IV. Miscellaneous

In a study[40] comparing the usefulness of liver scan, hepatic angiography, and peritoneoscopy in evaluating mass lesions of the liver, peritoneoscopy correlated with the final diagnosis 90% of the time, which was slightly higher than hepatic angiography and significantly higher than liver scan.

Peritoneoscopy has proved itself a useful tool not only in diagnosing hepatoma, but also and perhaps more importantly in establishing operability.[41]

In the evaluation of abdominal pain which might be due to adhesions this procedure has proved valuable.[42] With the patient awake and able to state whether manipulation of an adhesion reproduces the specific pain a secure diagnosis can be made and corrective surgery offered.

With the increased incidence of acute and chronic liver disease and the desire to evaluate long-term medical treatment by research protocol, peritoneoscopy with photographic documentation and directed needle biopsy of the liver allows the clinician to follow the gross and microscopic changes throughout the illness.

Clinical Value. In the Walter Reed study the clinical diagnosis was either confirmed or altered in 94% of the patients which is in keeping with the 95% figure cited in most reports.[43-46] However, the accuracy has been reported as low as 73%.[47] Others[35, 47-49] have approached the value of peritoneoscopy from the point of view of avoiding major surgery. Reports in the literature on this aspect vary from 50-70%. For example, in Horwitz' series[19] the clinical diagnosis proved accurate in only 52% of the cases. Peritoneoscopy allowed 43% of the patients to avoid laparatomy. Certainly there should be little argument about such a benefit to the patients with metastatic carcinoma who are at great risk from major surgery. If peritoneoscopy is viewed as an extension of the clinician's physical diagnosis and not as a replacement for laparotomy, internists and surgeons will agree on its usefulness in current medical practice.

Complications. There was no mortality in the Walter Reed review, which again is in keeping with the published low mortality rate of this procedure of 0.1% or less, i.e., comparable to that of simple paracentesis. Vilardell[50] reviewed the other complications attending peritoneoscopy and these are listed in table 2. In his series of 1,455 personal examinations, the complication rate was 1.2%. This figure rose to 2.5% when Bruehl[51] reviewed the complications reported by 67 physicians performing 63,345 peritoneoscopies. At Walter Reed the incidence of significant complications was 6%. This high complication rate is attributed to the fact that there were 16 physicians in various stages of training performing the 450 procedures. It must be stressed, however, that in only 5 of the Walter Reed patients (1%) was the hospitalization felt to have been prolonged by the complication.

TABLE 2

Complications of Peritoneoscopy

I. After Pneumoperitoneum:
  a. Shock
  b. Pain
  c. Dyspnea
  d. Subcutaneous emphysema
  e. Mediastinal emphysema
  f. Pneumothorax
  g. Scrotal emphysema
  h. Pneumocolon
  i. Puncture of stomach
  j. Air embolism
  k. Hemorrhage

II. After Peritoneoscopy:
  a. Pain
  b. Puncture of abdominal vein
  c. Puncture of epiploic vessel
  d. Puncture of epigastric artery
  e. Puncture of aorta
  f. Puncture of the liver
  g. Puncture of gallbladder
  h. Puncture of intestine
  i. Peritonitis
  j. Hepatic coma

III. After Biopsy:
  a. Hemorrhage
  b. Bile peritonitis
  c. Puncture of hydatid cyst

IV. After Cholangiography:
  a. Bile peritonitis

Conclusion

Peritoneoscopy is over 70 years old as a clinical technique and is of proven clinical value. Many reports since 1901 confirm the usefulness of the procedure when it is applied by the classic indications. Recent papers suggesting new indications support its expanding role in abdominal diagnosis and therapy. The mortality rate is low and the complication rate not excessive. Thus, the answer to the question posed in the title must be "yes." Peritoneoscopy has come of age.

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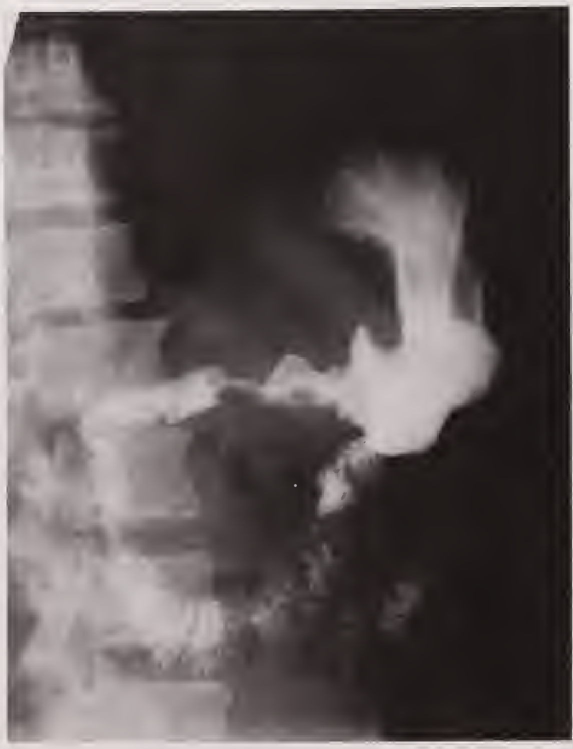
THE VIEW BOX

Prepared By
The Department of Radiology
Washington Hospital Center



This 51-year-old male presented clinically with vague abdominal pain and an approximate ten pound weight loss over the past two months. More recently, he described early satiety at meals but no decrease in appetite.
(For diagnosis see page 450)

The Current Status of Coronary Arteriography*

COLONEL MELVIN D. CHEITLIN, MC, USA, AND LT. COLONEL JAMES E. HASTINGS, MC, USA

The diagnosis of atherosclerotic heart disease has been and largely remains a diagnosis suspected on the basis of an accurate and compatible history. The definition of risk factors associated with this disease and the development of exercise stress electrocardiography have allowed us to strongly confirm or suspect this diagnosis. With the development of coronary arteriography we have gained a new and most valuable tool which has extended our ability to make or deny the diagnosis of coronary artery disease.

Concomitant with the development of more accurate anatomic diagnosis, several new surgical approaches to the problem of coronary artery obstruction have been devised.[1] The most exciting and promising of these is the saphenous vein bypass graft. This latter development has both broadened and confused the indications for coronary arteriography.

The purpose of this paper is to review the information we can expect to obtain from coronary arteriography and some of the pitfalls in interpretation that might be met, as well as to discuss the indications and contraindications for this procedure.

At the present time there are two generally accepted techniques for performing coronary arteriography. The first is the Sones technique, which utilizes a soft-nosed end-hole catheter introduced through a brachial arteriotomy.[2] The second is the Judkins' technique, which utilizes a pre-formed catheter advanced percutaneously from the femoral artery.[3] With both techniques cineangiographic recordings in multiple projections are made after contrast material is injected into each coronary artery. Each technique presents its own set of technical problems.

Interpretation of Coronary Arteriography

The fundamental requirement in the recognition of pathologic changes in the coronary tree is an understanding of the normal coronary artery anatomy and its variants. The normal and pathologic changes in coronary anatomy have been well documented by James[4] and Baroldi.[5]

The main left coronary artery arises from a single ostium, passes for a variable distance without branches until it divides into the left anterior descending (LAD) and the circumflex arteries (Figs. 1, 2). Other equally large branches called diagonal vessels may originate at or just distal to this bifurcation from the left anterior descending coronary artery (LAD) and course over the surface of the left ventricle. There are 1 to 3 of these. The LAD is situated in the interventricular groove and can be identified angiographically by the perpendicular relationship of the septal branches arising from it. It terminates around the apex of the heart in the posterior sulcus.

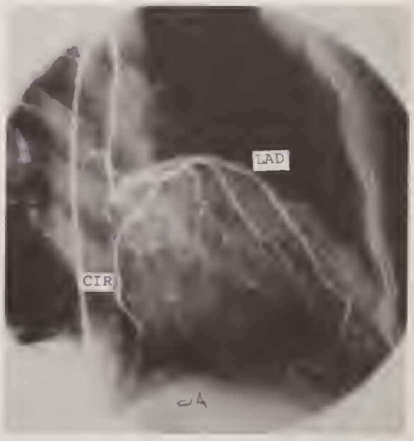


FIG. 1. Normal left coronary artery in RAO projection.

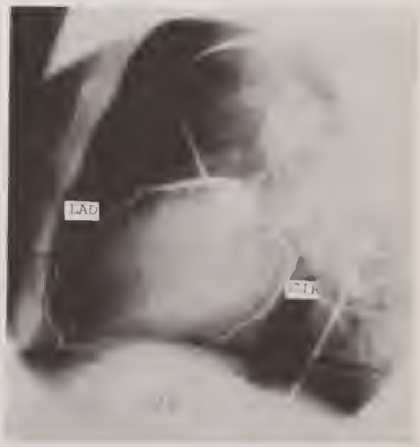


FIG. 2. Normal left coronary artery in Steep LAD projection.

The left circumflex artery (LCA) passes along the left atrioventricular groove, gives off marginal branches and terminates in the vicinity of the posterior descending coronary artery. It can be identified angiographically as lying in the bed of the coronary sinus.

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The right coronary artery (RCA) arises from the right sinus of Valsalva but frequently has 2 origins, the smaller of which is 1 or 2 mm from the larger and gives rise to the conus artery. The RCA passes along the right atrioventricular groove, giving off a large acute marginal and 2 or 3 smaller branches, and terminating when it gives rise to the posterior descending artery (Figs. 3, 4).

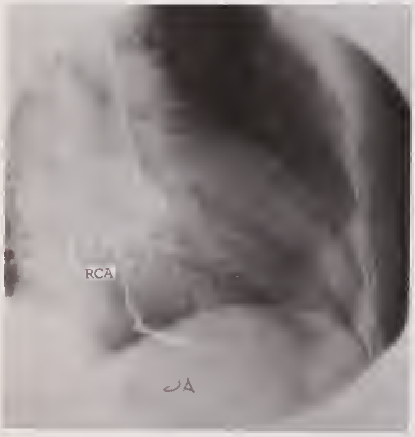

FIG. 3. Normal varient right coronary artery in RAO projection. Sharp angle of vessel gives the impression of a totally obstructed vessel. This is clarified when another view is obtained (Fig. 4).

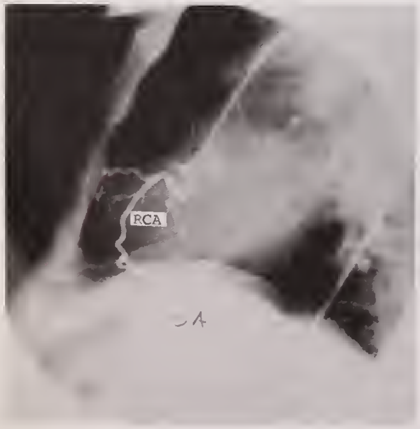

FIG. 4. Normal varient right coronary artery–LAD projection.

There is a reciprocal relationship between the LCA and the RCA. When the RCA gives rise to the posterior descending (90% of the time) it is termed "right dominant" and the LCA is smaller with fewer branches.[4] The 10% with a left dominant system and a very well developed LCA may have a very short tortuous RCA that is normal.

It is often difficult to see the origins of vessels at the point of branching. This is especially true at the bifurcation of the left coronary artery into LCA and LAD and the diagnonal branches. These vessels override each other, and discrete lesions can be missed. Narrowing of the coronary artery can be eccentric, so that the vessel viewed in 1 plane can look entirely normal, whereas a projection 90° to that view can show a severely obstructed vessel. Multiple views are required to separate these vessels for clear definition.

Ostial lesions are particularly difficult to define. The catheters have tapered tips that can pass beyond an ostial lesion. A good coronary angiogram should reveal a small amount of dye refluxing into the aortic root, thereby outlining the origin of the vessel. If an ostial lesion is suspected, an injection is made with the catheter not in the coronary artery but in its parent sinus of Valsalva.

Spasm of a coronary artery is a common phenomenon and can have the appearance of an obstructing plaque (Fig. 5). Spasm is fairly characteristic, however, in that it virtually always occurs at or just beyond the tip of the catheter. It is extremely common in the right coronary artery and unusual with the main left coronary artery, but more common if the tip of the catheter advances to either the LAD or LCA. The use of nitroglycerin before injections is helpful in abolishing spasm (Fig. 6). An injection later in the study or a sinus flush will usually clarify the situation, though rarely a study might need to be repeated to accurately define this situation. The presence of spasm stimulated by a catheter does not have clinical significance.

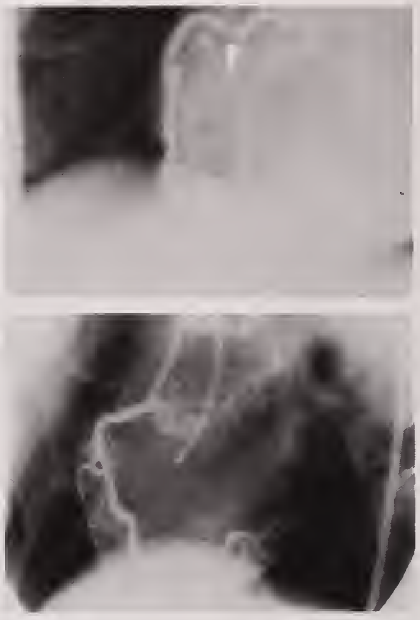

FIG. 5. Right coronary artery in LAD projection. Top frame shows spasm (arrow) just beyond tip of catheter. Lower frame is slightly different projection after nitroglycerin shows normal vessel diameter.

The length of the main left coronary artery is variable. With a short vessel the catheter tip can slip past the bifurcation, and a selective injection into the LCA can occur with the radiographic appearance of total occlusion of the LAD (Fig. 7).

In the RCA with 2 ostia, an injection into the lesser ostium would give the impression of a totally occluded main RCA.

With a vessel totally occluded there may be filling retrograde via collateral vessels. It is important to recognize the sequence of vessel filling or a total block could be missed since the vessel may fill in a retrograde fashion all the way back to the obstruction (Figs. 8, 9). Also, once a vessel is totally occluded and fills only by collaterals the caliber and the degree of pathologic involvement of the vessel is difficult to gauge. It may be considerably larger than its radiographic appearance would indicate because of the diminished volume flow of contrast.

Besides being able to see an obstruction on the coronary arteriogram, the film should be examined for evidence of collateral vessels. Schlesinger found collateral vessels in less than 10% of normal hearts.[6] In coronary arteriograms of 53 normal people or patients with non-obstructive lesions, Gensini and Bruto da Costa[7] found none with collateral vessels. The development of collateral vessels depends on regional myocardial hypoxia and diminished pressure beyond the obstruction. Their presence on the angiogram is excellent evidence of significant proximal coronary obstruction disease.

Accuracy of Coronary Arteriography

The question of how accurate coronary arteriography is in predicting pathologic obstruction of the coronary arteries must be raised. The major disease process we are concerned with is atherosclerosis of the epicardial coronary arteries, which involves vessels certainly larger than the (200 micra) size representing the resolution limit of good angiographic equipment.[8] Correlation of contrast studies with pathologic material has been carried out in several ways. Postmortem injection of contrast material followed by radiography and careful dissection have been performed.[9,10] Other studies correlate the premortem angiographic appearance with the postmortem dissection of the coronary arteries, although usually there is some time-lag between events.[11] Early studies suggested a poor correlation, but more recent evidence has been much more encouraging. Kemp and Gorlin's assessment of 145 vessels showed 23 errors (16%), but only 3 of these were functionally significant.[11] It seems clear that a perfectly normal angiogram is not associated with significant epicardial obstructive disease. Once a

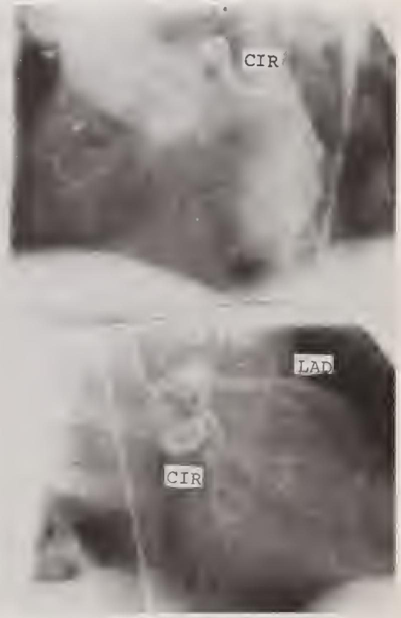

FIG. 7. Top frame is left coronary artery in LAD position showing opacification of circumflex vessel without LAD being seen. In RAO position (lower frame) both branches of main left coronary artery are visualized and are normal. This demonstrates isolated circumflex injection which could be interpreted as total occlusion of LAD. Prosthetic aortic valve is noted.

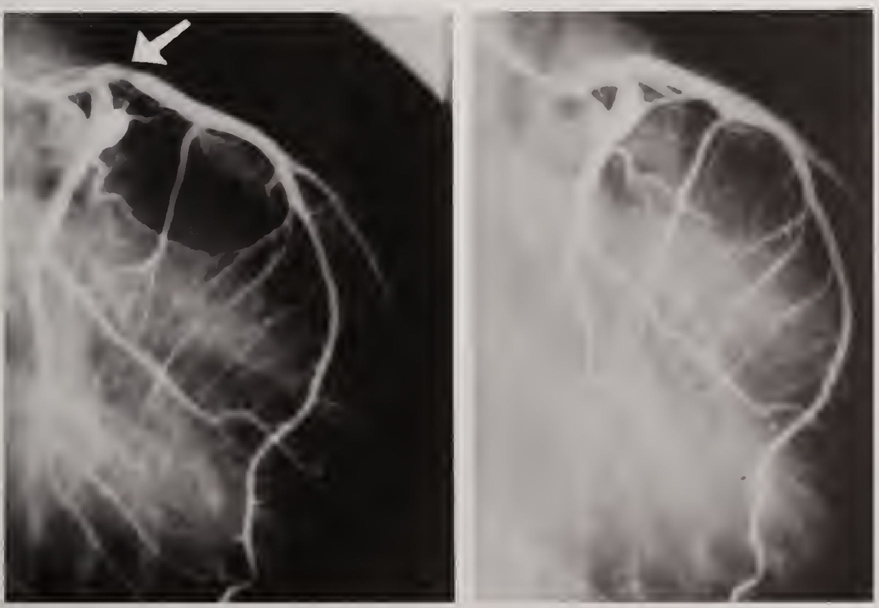

FIG. 6. Both frames show left coronary artery in RAO projection. Left frame shows narrowing of LAO just distal to catheter tip (arrow). Repeat injection after nitroglycerin showed this to be normal.

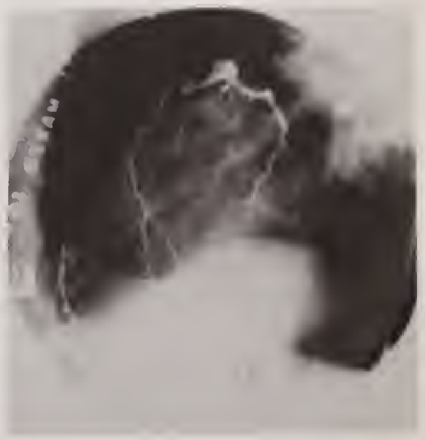

FIG. 8. LAD projection of left coronary artery shows almost normal appearing vessel (see Fig. 9).

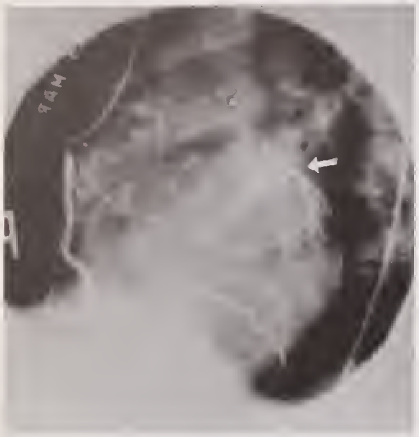

FIG. 9. LAD projection of right coronary artery showing retrograde filling of a totally obstructed branch of the circumflex (arrow) not suspected from left coronary artery injection.

plaque is present and recognized, the tendency is to underestimate the degree of obstruction.

Two problems present themselves. First, what are the possibilities for missing coronary disease with a normal technically adequate coronary angiogram? Second, how does one assess functional significance of the plaque when seen?

A number of patients have been described with anginal pain and a positive stress electrocardiogram with "normal coronary angiography". We have seen several people who have had classic clinical myocardial infarctions and 3 to 6 months later have normal coronary arteriograms. Several explanations have been advanced to explain these phenomena:

1. There may be a missing diagonal or other minor branch not visualized because of a lesion at the origin of the vessel.

2. The symptom-complex may be secondary to involvement of coronary vessels too small to be seen on the arteriogram.

3. There may be another cause other than an anatomic abnormality resulting in a discrepancy between supply and demand for coronary blood flow such as the high-wall tension of aortic stenosis or the short diastolic flow-period of the tachyarrhythmias. There may be other purely physiologic abnormalities which limit the ability of the blood to deliver oxygen to the myocardium such as the recently publicized abnormality in oxyhemoglobin dissociation.[12]

4. There may be a reversible obstructive phenomenon, such as a thrombus that has undergone fibrinoysis or coronary arterial spasm.

Dr. James[13] has suggested that the usual explanation for these discrepancies between pathophysiology and arteriography is inadequate technique resulting in failure to detect a major vessel obstruction. Small-vessel involvement as the cause of the discrepancy is not really tenable when, as Baroldi and Manion showed,[14] very extensive small-vessel disease as is seen in thrombotic thrombocytopenic purpura is not characterized by angina or significant myocardial fibrosis.

Our present opinion is that, with proper technique, if there is no lesion demonstrated in good quality films in multiple views, if there is no evidence of collateral development, if all the major vessels are present, then we can conclude that there is no significant epicardial coronary arterial obstruction.

The second problem in interpretation is the assessment of the functional significance of the plaque once identified.

Enos,[15] in an autopsy series on young men killed in Korea, found a 10% incidence of severe single-vessel disease, all patients presumably having been asymptomatic. The finding of a plaque does not necessarily mean decreased coronary blood flow and symptoms. A 50% reduction in diameter reduces the cross-sectional area of the vessel by 75%. This is the level of an obstruction at which a pressure gradient is created.[16] It is at this point where there begins to be a diminution in blood flow at times of increased physiologic demand. Just as "ischemic" ST-segments on the stress test and myocardial lactate production are the physiologic and biochemical analogues of diminished coronary flow denoting a significant plaque, so the presence of collateral vessels is the anatomic analogue.

Indications for Coronary Arteriography

Were coronary arteriography a benign non-invasive technique without cost in terms of mortality, morbidity, expense or professional time, the ubliquitous nature of the disease we are dealing with might dictate that all patients over the age of 35 years would be candidates for coronary cineangiography to ascertain the presence of the disease and its severity. A slightly less expansive view presently held by many centers is that any patient in whom the diagnosis is entertained should have this diagnostic evaluation. In order to examine this position, we must know the cost to the patient, in terms of morbidity and mortality, and then weight this against the possible benefits accrued.

The mortality published by Sones[8] and Judkins[17] for the procedure is 1 to 2 per thousand studies. Complications occurred in 2% of 3,000 Sones' procedures.[18] In a Co-operative Study of Cardiac Catheterization in 1968, complication rates of 6% were listed from hospitals other than the Cleveland Clinic.[18] In the San Francisco Bay area a canvas of hospitals revealed a mortality of 1 to 2%.[19] In another Western City having 4 large institutions including a medical school, the mortality was between 2 and 3.7%.[19] The morbidity was closer to 6% than 2% and morbidity from acute myocardial infarction alone, if all changes in electrocardiogram and enzyme studies are included approaches 3%.[20] Suffice it to say that coronary arteriography may be more hazardous in many centers than would appear from the reports of large groups. The reason for this is unclear. Experience in performing thousands of studies certainly should decrease morbidity and mortality. In the large series, 30 to 40% of the patients studied have had normal coronary angiograms and, at least in our experience, our morbidity has usually been in patients with severe coronary disease. The larger number of normal studies in which the chance of problems is least would further decrease the total morbidity and mortality of coronary arteriography.

Benefits of Coronary Arteriography

Weighed against the cost are significant benefits for the patient that accrue from the information obtained. This is especially true in the following situations:

1. Patients who are diagnositic problems. Patients who have atypical chest pain with or without coronary-risk factors and who have been labeled by their physician or even themselves as having coronary artery disease represent a clear indication for investigation. The diagnosis of coronary heart disease carries profound economic and psychological penalties, and the presence of a normal coronary arteriogram is powerful objective supporting evidence of the absence of coronary disease.

Another group of patients in this diagnostic category would be those who by virtue of an abnormal electrocardiogram have been diagnosed as having had an "asymptomatic" myocardial infarction. In the past the development of a conduction defect such as right or left bundle-branch-block has often been attributed to coronary arterial disease. It now appears that the minority of totally asymptomatic people with left bundle-branch-block have coronary artery disease as the cause.[21]

"Ischemic" ST-segment changes on stress electrocardiography do not completely correlate with abnormal coronary angiography. Indeed, in some series the incidence of false "positive stress tests" may be as high as 31%.[22] Martin and McConahay[23] at Letterman General Hospital correlated the results of maximal treadmill tests in 100 patients with coronary arteriography and described a "false positive" test of 1.0 mm "ischemic" ST-depression in 8%. This is probably a more realistic figure.

2. Patients with angina and valvular heart disease in whom surgical treatment is being considered. This question usually arises in aortic stenosis, but aortic valvular insufficiency as well as mitral valve disease can also present this problem. Coronary arteriography is helpful in assessing the risks of surgery. The risk of prosthetic valve replacement is greater with, than without significant coronary disease.[24] In the presence of combined aortic-valve disease and significant coronary arterial obstruction some surgical centers today are carrying out the combined operation of valve replacement with "jump" bypass graft. If the patient has mild to moderately severe aortic stenosis, the coronary angiogram can define the presence of the disease causing the patients' symptoms and perhaps alter the indication for surgery.

3. Patients who are being evaluated as candidates for coronary artery surgery. This group represents the largest and also perhaps the most controversial group at the present time. If an ideal operation were available at low risk, with results which were known over a period of years, this would no longer be a problem. At present the facts are these:

There are much statistical data available on patients with angina and myocardial infarction and what happens to them in 5, 10 and 15 years.[25-27] In the individual case, however, we are unable to predict the course of the disease.

We do not know the natural history of angiographically proven lesions. Ross and Friesinger[28] followed a group of patients after coronary angiography without surgical intervention. Surgical statistics were analyzed over a 7-year period. People with angina pectoris have a better prognosis than those with myocardial infarction. The more important finding was that those with mild to moderate disease as evidenced by single-vessel disease had the same mortality rate as patients without coronary artery disease. The patients with single-vessel disease have been thought to represent the best surgical candidates. It appears that they may do even better without surgery.

A significant contribution to the natural history of severe proximal coronary disease has been made by Moberg et al[29] They followed 200 patients after coronary arteriography for up to 9 years. Their findings were that single-vessel anterior descending disease has a 4% yearly attrition rate and single right coronary or circumflex lesions demonstrated a 1.8% yearly death rate. Patients with 2-vessel disease had a 44% 7-year mortality rate and with 3-vessel disease a 70% 7-year mortality rate. It appears that survival is related to the number of vessels involved.

The natural history of the saphenous vein-graft is not yet known. Some facts are available. Mortality for the surgery is 6-10%, and those with poor ventricular function have a mortality of 20-25% or higher.[30] Recent studies have suggested that the incidence of myocardial infarction occurring with or shortly after the surgery is as high as 10% and may be higher.[31] Up to 90% of the grafts are patent on re-study within weeks, but graft occlusion at 1 to 3 years may be as high as 20-30%.[32] Poor run-off is a significant problem, and if flow is less than 20 cc/minute almost all grafts will close presumably from thrombosis at the site of anastomosis.[33] The adequacy of the run-off bed is difficult to evaluate from the initial angiography and is only evident after the graft is in place. Grafts will also close late from subintimal fibrous hyperplasia, and we have seen this in a case as early as 3 months postoperatively.[34] The magnitude of this problem is as yet unknown. Once the saphenous vein-graft is successfully placed there is evidence that collaterals decrease in extent and in many the severely obstructed bypassed coronary vessel now goes on to complete occlusion.[35] The perfused myocardium is now completely dependent on the patency of the graft. If the graft is placed distal to several plaques there may be poor proximal perfusion, and many major vessels such as diagonals and septal perforators are still poorly perfused. There is as yet no adequate solution to this problem.

Conclusions

Our indications for coronary angiography are based on the information we have presented. At the present time we study patients in the following categories:

1. Patients with chest pain with atypical features for angina when the diagnosis is in doubt. In this case the study will clarify the diagnosis and contribute to the management of the patient's problem.

2. As part of a pre-surgical evaluation when the decision has been reached for surgical intervention based on continued severe angina in spite of good medical management. This management includes control of weight, blood pressure, and cessation of smoking cigarettes as well as the use of long-acting nitrites and beta-blocking drugs, and the vigorous treatment of even minor degrees of congestive heart failure. In our experience it is very unusual to perform coronary angiography and have our surgical colleagues unable or unwilling to attack the pathology demonstrated. By confining our studies to patients pre-selected for surgery by their clinical status, we avoid the problem of recommending surgical intervention on the basis of anatomy alone, a position that is difficult to justify with our present information, i.e., this operation has not been shown to be prophylactic in minimally symptomatic patients.

3. In the investigation and management of patients with crescendo preinfarction angina. In various series, myocardial infarctions occur 20 to 40% of the time within 1 year in people with crescendo angina, especially if the angina continues at bed rest.[36,37] It is possible that "prophylactic" surgery is justified here.

4. In the young patient (under 35) with angina and/or a myocardial infarction, even without incapacity, coronary angiography is helpful prognostically in defining the group with minimal or absent coronary artery disease and those with localized disease. It appears that these people have a better prognosis than those with diffuse coronary disease.

If the diagnosis of coronary heart disease is highly probable, with classic angina pectoris or a definite myocardial infarction, and if the patient's condition is stable, non-progressive are not incapacitated on medical management, coronary arteriography is not indicated.

Coronary angiography in the face of a recent myocardial infarction may have increased risk associated with it. One of the present controversial situations which is actively being investigated is the role of coronary angiography and surgery in the presence of an acute myocardial infarction with shock. It is possible that a better salvage will be obtained with than without surgery.[38]

We have avoided discussion of anatomy per se as being an indication for surgery. It is generally agreed that certain anatomic situations are unusual in analyzing large numbers of coronary angiograms. It is probable that these lesions are unusual because they are associated with a high mortality rate. Two examples of this situation are lesions proximal to the first septal perforator in the left anterior descending coronary artery and total occlusion of the main left coronary artery. Another "critical" anatomic situation would be the presence of a completely occluded vessel filling distally by collateral vessels from another coronary which itself has a high-grade proximal obstruction (Fig. 10). In this case a very large area of the left ventricle is dependent on blood flow past a critical obstructive lesion.

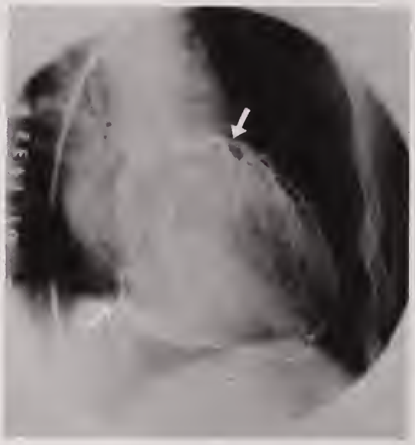

FIG. 10. Left coronary artery injection in RAO projection showing moderate obstruction of LAD (arrow) and retrograde filling of right coronary artery (arrow) denoting total obstruction of vessel.

These situations are taken to be indications for surgical intervention in a number of centers even in the relatively asymptomatic patient. At the present time we do not believe that the natural history of either the lesion or the grafts is known well enough to justify this recommendation.

Finally, coronary arteriography is indicated in the follow-up evaluation of patients with coronary by-pass grafts. Filling of the graft is not proof

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that the flow through the graft is adequate. The functional success of the graft will depend not only on the patency of the graft but also on the lack of symptoms and the evidence of decreased ischemia by objective studies such as stress electrocardiography.

It is not our desire to 'down play' the importance of the recent advances in coronary artery surgery. There is no question that after many false starts the development of the microsurgical techniques that make coronary bypass possible is most promising. The initial subjective and objective results in patients with severe angina after surgery is even more impressive. In our enthusiasm we must avoid the danger of subjecting our patients to unnecessary hazards before we know that the danger to their lives and hearts is greater without than with surgery. Until we have this evidence, in our opinion it is better to be conservative in recommending coronary arteriography and to reserve surgical intervention for those most likely to be at a risk in the near future.

Coronary arteriorgraphy is a technique which allows us to know much about the anatomic condition of the coronary arteries. The indications for coronary arteriography are basically to properly advise therapy for the patient. As surgery improves and we learn more about the natural history of the vein grafts, the indications for coronary arteriography will change.

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Acalculous Cholecystitis—A Possible Complication of Parenteral Hyperalimentation Report of a Case*

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Since 1968 when Dudrick[1] introduced parenteral hyperalimentation (PHA) as a means of maintaining or restoring nutrition in severely ill patients who are unable to take nutrition by the oral route, the procedure has found widespread application. The technique of PHA is currently used in preoperative preparation of malnourished patients, the therapy of the patient with gastrointestinal fistulae, chronic bowel obstruction, or one who is malnourished because of inflammatory bowel disease. It is now also being used by some investigators as therapy for acute renal failure,[2] and its possible utilization in the therapy of reversible hepatic failure has been proposed.[3]

Because PHA requires an indwelling subclavian catheter for long-term infusion of hypertonic glucose solutions, many complications such as pneumothorax, septicemia often due to a fungal organism, and hyperosmolar coma with dehydration have been reported. Other complications have occurred and have been detailed in a recent review.[3] Not included in that list is the occurrence of acalculous cholecystitis. This report describes a patient with extensive regional enteritis who developed acalculous cholecystitis following the use of PHA.

Report of Case

S.G., a 22-year-old white man first developed cramping lower abdominal pain, diarrhea, and weight loss in 1968. Barium enema revealed enteritis involving the terminal ileum and the cecum. He failed to improve on medical therapy. Resection of the distal 14 inches of the terminal ileum plus the cecum with end-to-end ileocolostomy was then accomplished in September, 1969. At surgery the gallbladder was noted to be normal in its appearance. Postoperatively the patient did well and gained weight from 110 lb. to 130 lb. However, in February 1970 he noted recurrence of cramping lower abdominal pain, diarrhea, nausea, vomiting, anorexia, and weight loss. Therapy with Azulfidine and Lomotil was instituted. Prednisone, 30 mg daily was added in September 1970. An oral cholecystogram performed at that time was normal. Severe malnutrition and continuing abdominal symptoms prompted hospitalization in December 1970 for PHA. Upper GI series demonstrated changes within the duodenum, jejunum, ileum, and ascending colon compatible with regional enteritis. Serum amylase was 100 units/100 ml (normal less than 160).

A subclavian catheter was inserted, and a 30-day course of PHA was begun. During this period the patient became asymptomatic with cessation of the abdominal pain and a weight gain of 25 lb. PHA was then discontinued, and gastrointestinal X-ray studies demonstrated marked improvement, manifested by a decrease in thickness in the duodenal and jejunal folds. When oral intake was resumed, the patient complained of right upper quadrant pain occurring in the postprandial period. He was discharged home on no medication but returned 3 days later complaining of severe right upper quadrant pain, fever, and chills. He weighed 125 lb. The pulse rate was 140/minute, the blood pressure 110/80, and the rectal temperature 101° F. There was marked tenderness in the right upper quadrant with localized rebound. Bowel sounds were diminished. No masses were palpable. Rectal examination was normal.

Laboratory data: WBC 17,800 with 85% neutrophils and 7% bands; hematocrit, 34.8%; amylase, 80 units/100 ml.; total bilirubin, 0.3 mg. %; direct, 0.2 mg. %; SGOT, 21 units; SGPT, 21 units; alkaline phosphatase, 7.5 King-Armstrong units; BUN, 9 mg. %. Chest, flat and upright films of the abdomen disclosed no abnormality. Intravenous fluid and nasogastric suction were begun. Ampicillin, 500 mg, was given intravenously every 4 hours. No improvement occurred in the ensuing 8 hours and a laparotomy was performed. An acutely inflamed gallbladder with adherent omentum was found. After removal, the gallbladder was opened. The wall was thickened and the lumen was filled with thick black inspissated bile. No gallstones were present. The common bile duct was not explored. Palpation of the head of the pancreas disclosed no evidence of pancreatitis. Inspection of the bowel revealed no signs of active regional enteritis. Subsequent microscopic examination of the gallbladder revealed acute cholecystitis (fig. 1). Edema of the mucosa and submucosa was noted. The wall of the gallbladder averaged 0.5 cm. in thickness. There was infiltration by polymorphonuclear leukocytes. No abscess formation was found. Serosal thickening and hyperemia were present. The absence of any calculous formation was confirmed.

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Postoperatively the patient developed transient hyperbilirubinemia which resolved over a period of 5 days. He remained asymptomatic and was discharged on the 10th postoperative day.

Discussion

Although not a common disorder, a-calculous cholecystitis has recently received considerable attention. Most cases have occurred during the early postoperative period in elderly individuals following major surgery. Several authors have described the occurrence of a-calculous cholecystitis in young males after severe injuries[4] and in patients with massive burns.[5] The common feature in all reports is the occurrence of the disorder in the early postoperative period. The predisposing factors noted by various investigators have been the presence of fever, dehydration and/or overwhelming septicemia, as well as the administration of narcotic analgesics.

It is of note that most such patients have been maintained without oral intake for prolonged periods of time. In several reports[4,5] symptoms have developed after oral intake was resumed. Despite the variation in theories of pathogenesis, gross and microscopic pathologic findings have been characteristic. Gross evidence of acute inflammation characterized by serosal hyperemia and adherence of the omentum to the gallbladder is usually evident to the surgeon. The gallbladder wall is thickened, and the lumen contains thick inspissated bile. Stones are not present. In many cases, culture of the gallbladder contents, particularly in those cases associated with septicemia, demonstrated bacteria.[5]

The occurrence of acute acalculous cholecystitis in our patient with neither a history of recent surgery or any severe injury suggests another mechanism for the development of acute acalculous cholecystitis. The possibility that the disorder may be influenced by "sluggish bile flow" has been mentioned in other reports[4] but has generally been rejected. Most reported cases were believed related to the occurrence of septicemia and/or dehydration. However, altered bile production and flow may well be important pathogenetic features, particularly in the patient receiving PHA.

It is well known that in the absence of any oral intake, gastrointestinal secretions are diminished. In addition, there is experimental evidence that the secretion of upper gastrointestinal juices is further diminished by PHA. Hamilton et al[6] reported a decrease of 50.5% in the volume of duodenal fluid in dogs undergoing hyperalimentation. The bilirubin content of these duodenal secretions was diminished by 86.2%. It is known clinically that patients with gastrointestinal fistulae demonstrate prompt reduction in flow from such tracts when placed on PHA.

Because of the experience gained in this patient, 2 additional patients with regional enteritis in whom PHA was planned underwent oral cholecystography prior to the institution of therapy. In both a normally functioning gallbladder was demonstrated. Oral cholecystography was repeated in both after 10 days of PHA. The gallbladder was not visualized in either on 2 successive days. However, after a fatty meal was given and the study repeated, faint visualization of the gallbladder was obtained.

These observations suggest that biliary secretion and gallbladder emptying are diminished during PHA. Continued re-absorption of water from the stagnant contents of an otherwise normal gallbladder, results in inspissation of bile. When the patient resumes eating it is likely that the gallbladder is emptied uneventfully despite its viscid contents. In our patient it appears that the gallbladder was unable to empty when oral intake was resumed and cholecystitis resulted.

There is a dramatic increase in the incidence of cholelithiasis in patients with regional enteritis. In a recent report this incidence was 34%. It has been suggested that this high frequency of cholelithiasis is due to malabsorption of bile salts from the gut because of damaged or resected terminal ileum. This, in turn, results in a diminished bile-salt pool which has been associated with an increased incidence of cholelithiasis. However, acalculous cholecystitis has not been associated with regional enteritis.

Pancreatitis may occur in the presence of regional enteritis, probably due to pancreatic duct obstruction when duodenal involvement is present.[8] In the presence of pancreatitis acalculous cholecystitis might also be anticipated. Multiple serum amylase determinations were normal in this patient and at surgery no pancreatitis was found.

Acalculous cholecystitis, as a complication of PHA, is probably rare, since it has not been reported to date, despite the wide use of PHA. However, this potentially serious complication may be prevented by the administration of periodic fatty meals or possibly cholecystokinin.

Of equal significance is the fact that acalculous cholecystitis may occur in clinical settings other than those which have generally been recognized, i.e., the postoperative state, following severe trauma, or after generalized burns. Since it is likely that failure of the gallbladder to empty during PHA led to acalculous cholecystitis in this patient when oral intake was resumed, it seems reasonable to conclude that attempts should be made periodically to empty the gallbladder in patients in whom oral intake is prohibited for prolonged periods of time.

Summary

The case of a patient with widespread regional enteritis who developed acalculous cholecystitis after parenteral hyperalimentation (PHA) is reported. It is probable that PHA results in a decrease in gallbladder emptying as well as diminished bile flow. It is suggested that the gallbladder became filled with a concretion of viscid bile which could not be emptied when the patient resumed oral intake. This potential complication of PHA can be averted by the periodic feeding of a fatty meal.

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THE VIEW BOX

(from page 440)

DIAGNOSIS: Adenocarcinoma of stomach

The mucosa in the antrum is destroyed and there is submucosal infiltration along the lesser curvature to the cardia. There was involvement of the pancreas, the regional lymph nodes and the liver by tumor. A palliative surgical procedure was performed. There was no involvement of the duodenal bulb and this is true for most cases. Apparently the pylorus acts as a natural barrier to the spread of tumor.

Carcinoma accounts for about 60 per cent of localized infiltrating lesions of the stomach. Other possibilities include granulomatous diseases such as sarcoid, lues, tuberculosis and certain fungal disease. Infrequently regional enteritis, hypertrophic gastritis of the antrum and lymphoma can produce a similar appearance.

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Mitral Annulus Calcification in Marfan's Syndrome
Report of a Case*

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Isolated calcification of the mitral valve annulus in elderly patients without X-ray or pathological evidence of leaflet involvement is a well reported entity.[1] Generally, it is of no clinical significance. However, it may be associated with mitral insufficiency or stenosis of severe degree and/or conduction disturbances.[2]

Annular calcification has been also noted in cases of rheumatic mitral disease but is associated with X-ray and/or pathological evidence of leaflet calcification. Clinically these patients are indistinguishable from other mitral-rheumatic patients.

In his 1966 monograph McKusick[4] described mitral annular calcifications as an associated finding in Marfan's syndrome, and 2 other cases have since been reported. We report another patient with Marfan's syndrome and isolated mitral annular calcification.

Report of Case

M. M., a 28-year-old white woman, was referred to Walter Reed General Hospital in October 1970 for evaluation of a heart murmur first heard at the age of 8. Though the patient was asymptomatic at that time, her activities were restricted until the age of 18, when she was told that the murmur "had almost disappeared". The patient remained asymptomatic after resuming normal activities until 2 years prior to admission when she noted marked shortness of breath, dyspnea on exertion, and orthopnea while camping at 9,000 feet. She had no history of pedal edema, palpitation, chest pain, or syncope.

The family history is negative with respect to heart disease, sudden death, or heart murmurs. The patient states that her mother is tall, thin, double-jointed, and wears glasses.

The patient's medical history and review of symptoms reveals 2 operations at the age of 9 for severe scoliosis and a need for glasses since high school, though vision is not corrected to normal with them.

Physical Exam: Height 63.5 inches. Upper/lower ratio 28.25/35.25 inches (ratio 0.76). Weight 101 lbs., BP 124/86, pulse rate 126/min., and temperature 98°F orally. Arm span 74.75 inches. The patient was a well-nourished, thin white woman in no distress. Slit lamp exam revealed bilaterally irregular lenses with a superior-anterior displacement of the left lens. The mouth showed a high-arched palate. The chest showed mild pectus excavatum and a marked rightward scoliosis. The cardiac apex was in the fifth left intercostal space in the anterior axillary line with a systolic thrill maximal at the apex. S1 was soft, S2 was obscured by a 5/6 holosystolic regurgitant murmur loudest at the apex with radiation to the axilla but easily heard over the entire precordium, neck, and back. No diastolic murmurs were noted. There were long, tapered fingers which were hyperextensible. The thumb could be apposed to the forearm. The remainder of the physical was unremarkable.

Laboratory Data: The CBC, BUN, creatinine, uric acid, calcium, phosphorus, bilirubin, SGOT, LDH, total protein, albumin, and cholesterol were all within normal limits. A urinalysis was normal. The serology was nonreactive. Chest X-ray films showed the prominent S-shaped scoliosis and over-penetrated views of the chest showed ring-like calcification in the heart in the area of the mitral annulus (fig. 1). The electrocardiogram was normal.

Right-heart catheterization was performed: Mean right atrial pressure 2mm Hg with an a wave of 4 and a v of 3; RV pressure 20/0-4; pulmonary artery pressure 20/10 with mean of 15; pulmonary artery wedge 10 with a V of 16mm Hg. Pulmonary artery appearance of hydrogen was 12 seconds. These findings were consistent with clinically insignificant mitral insufficiency.

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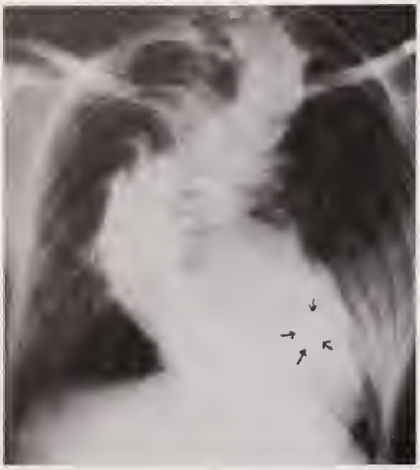

FIG. 1. Marked scoliosis and circumferential calcification of the mitral annulus (indicated by arrows).

Discussion

From our case and the 2 other cases summarized in table 1 it appears that there may be some common characteristics: 1. A grade 3/6 or greater apical systolic murmur with radiation to the axilla. 2. Apical systolic thrill. 3. Age below 30. 4. Calcification easily seen on plain films of the chest. 5. Catheterization data showing evidence of mitral insufficiency.

Our patient had clinically insignificant mitral insufficiency on the basis of history, catheterization data, and absence of left atrial enlargement or left ventricular hypertrophy on both ECG and chest X-ray film. Physical examination, however, would have suggested severe mitral regurgitation. An abnormality of the mitral leaflets or chordae was not ruled out in this patient because of reluctance to do a left-heart study in a patient with probable aortic-root disease.

This patient and the others cited represent another abnormality of the mitral valve apparatus in Marfan's syndrome. However, we are unable to say if the calcification in the annulus contributed to or was incidental to mitral insufficiency.

TABLE 1: Pertinent Cardiac Data on Other Reported Cases

Reference	5	6
Age	24	21
Sex	M	F
Symptoms	Easily fatigued	None
Cardiac examination	PMI at AAL, accentuated P2, fixed split S2, 3-4/6 apical murmur.	PMI 3 cm. left of midclavicular line apical systolic thrill. 5-6/6 palsystolic murmur at apex, disatolic rumble at apex.
Electrocardiogram	P mitrale, left atrial enlargement, left ventricular hypertrophy.	Frequent pACs, left ventricular hypertrophy.
Chest X-ray films	Left atrial and ventricular enlargement and annular calcification.	Left atrial enlargement, left ventricular hypertrophy, linear calcification of mitral annulus.
Catheterization data	Normal left- and right-heart catheterization, left ventricular cine showing mild mitral insufficiency.	Normal left and right catheterization, but wedge tracing suggests mitral insufficiency; left ventricular cine showed severe mitral insufficiency and left atrial enlargement.
Remarks	Had prosthetic mitral valve, but, no demonstrated abnormalities of mitral valve.	

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HMO's and Other Delivery Systems*

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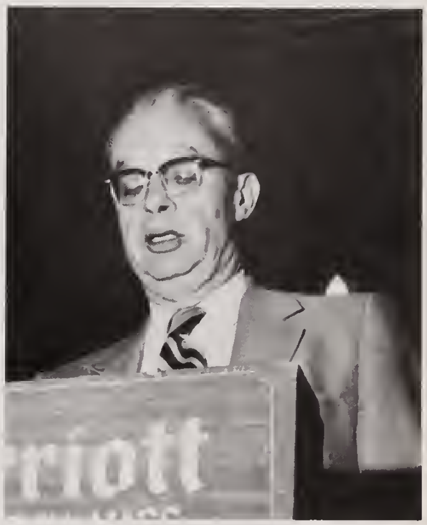
The physicians of this country hold to the standard of good medical care for all citizens. It is the method by which this care can be delivered and reasonably financed that is causing so many varied approaches to be suggested. The American Medical Association very clearly has stated time and time again that it endorses and supports a multiplicity of delivery methods for health care. It strongly favors a pluralistic system since it is felt that even within a single area economic, geographic, ethnic, cultural factors will call for different approaches to different segments of the population.

On October 25, 1971, speaking at an open meeting in the State of Maryland, Gilbert R. Bernhart, Ph.D. of the Department of Health, Education and Welfare spoke on the subject of Experimental Health Service Systems. His presentation was theoretical as to what might be called the ideal type of system toward which efforts should be bent. His points were outlined as follows: 1) *Criteria*—under this a) there must be free and easy *access*, b) uniform *quality*, and c) *economy* of service; 2) *Method*—meaning there must be identification of and way of keeping track of the population by a) enrollment in groups or centers, and b) assignment of population; 3) *Organization*, including a) there must be standards and norms of care, and b) distribution of care by assignment if necessary of personnel by need; 4) *Financial controls* by corporations utilizing labor, management, consumers and so on.

It is necessary to reiterate that these points comprise what was presented as the ideal situation. If we focus for this discussion primarily on the first—the criteria, there is a base from which to work. These goals then are: a) access to the system for all needing health services, b) reduction of costs by workable controls, and c) quality care based on standards or norms.

It is my intent to put before you a few points, which while not directly related to the subject of Health Delivery Systems including HMOs, will have some bearing. The total number of physicians in the U.S. as of December 31, 1970 was 334,028 of which 278,535 or 83.4 per cent were in patient care. As a primary activity, medical teaching took 1.7 per cent, administration 3.6 per cent, research 3.6 per cent, miscellaneous 0.8 per cent and 6.9 per cent are missing or retired. In 1960 there was one medical doctor for every 712 people while in 1970 there was one for every 632 patients or potential patients. Group practice in the last year surveyed (1969) accounted for 40,000 physicians in 6,371 groups. Of these groups, 49.7 per cent were single specialty, 12.3 per cent were general practitioners, and 38 per cent were multispecialty groups. However, the medical community is changing. A number of



physicians nearing 60 years of age are tired of paper work, of malpractice problems, of wrangling with intermediaries or with patients who believed it when they heard 100 per cent of their medical care was covered. They have stopped practice and have gone to work in homes, industry, or groups and thereby have reduced the number of hours they work. A second group of younger physicians is looking for what industry has; namely a 40 hour week, paid vacations, a pension or retirement plan, sick leave and other benefits. In addition there is a disinclination of a physician to practice in rural or slum or low income neighborhoods for several reasons: 1) he often lacks contacts with other physicians (contact stimulates a man to continue learning), 2) he does not feel his family has an adequate life including participation by him, and 3) he has difficulty making a decent living. Since most physicians' income in many areas now comes through

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Title 18 or 19 there is, as you are well aware, a trigger point of $25,000 gross income from these governmental funds at which payments cease and investigation is launched. This arbitrary figure is a gross figure and in no way takes into account the hours worked, the charges made (often under the allowed amount) or the expenses incurred. So while access to health care is available, it often takes quite a bit of doing to get there. One of the primary points to be remembered is that while in 1969 there were in the private field 7,144 hospitals offering 1,649,663 beds and of these 7,144 hospitals 5,853 were community hospitals, there was not a hospital located in every hamlet in the United States or in every section of every city in this country. So, as with hospitals, we do not have uniform distribution of physicians. How to get these men into areas of agreed upon need? Several alternatives have been raised such as substitution for military service, incentives of the subsidization type, compulsion possibly by extension, and loans when coupled with the Student American Medical Association Medical Education Community Orientation Program whereby students are sent into the rural or community areas for 3 months exposure may also help. These are to be kept in mind as the approach is made to our main subject.

HMOs

Ever since then Undersecretary John Veneman of HEW first used this term on March 23, 1970 it has become a catch phrase and while at first limited in its thoughts to Title 18 beneficiaries, it has now been applied to the entire population. It is a concept which has been properly identified as an experimental method of delivery care. There exists really no concrete definition of an HMO and legislative proposals in this subject offer a variety of conditions which, in the opinion of the sponsors of the bills, adds something to the picture. Basically, it appears, the HMO is a corporation which contracts with persons (known as enrollees) to furnish them comprehensive care including treatment and prevention on a per-capita annual basis. It is to furnish medical care through physicians who are either partners with or employees of the corporation. Such an HMO stresses access to care, good quality care, and attempts to combat the cost rise factor by physician incentives based upon utilizing the least expensive services which are appropriate to the patients needs. Two factors raise themselves here: 1) the utilization of medical manpower in examining or counselling with the well or near well will further stretch the care of the sick, and 2) while the theory of bringing persons in for a regular check-up (such as an automobile undergoes) sounds attractive, there is not yet a proven cost-to-benefit ratio that has been determined anywhere. So the basic theme of the proponents of immediate implementation of the HMO concept has become that early diagnosis and health maintenance will result in long range savings in the future. It is important to realize data collection involving the cost benefit ratio is underway with the funding at $6.5 million of HEW money in 1971 of 35 planning grants, 16 feasibility grants and 15 research and demonstration projects—a total of 66 HMO grants and contracts. These, when completed, should give all of us a better idea of whether this concept will be worthwhile. In strictly interpreting HMOs and their functions it must be clear that any organization of this type must be able to deliver or provide all reasonable services to those enrolled and to provide these services, including hospital care, at a reasonable cost. It seems imperative then that this HMO must be able to insure access to hospital facilities to its people and to its medical staff. Therefore of the different programs as of now in the country, it appears the Kaiser-Permanante which is always used as the prototype, as well as the few remaining railroad hospitals and union plans as the United Mine Workers Plan, might easily qualify. They have integration of professional and institutional services. The next most readily adaptable to an HMO would probably be a medical school-teaching hospital combination or a hospital based corporation utilizing the medical staff or portions thereof needed. The hospital-physician inter-relationship is most important. While fully recognizing this treads into the proposed Ameriplan of the American Hospital Association, it can only be stressed the ultimate liability for services and their quality presents a problem for the corporation which must be carefully evaluated. In the areas of Washington, D.C. both George Washington University School of Medicine and Georgetown University are attempting to activate HMOs for certain segments of the population in geographic areas of the community. Existing pre-paid groups which don't have a relationship or control hospital services by which they can review such things as cost and availability are a step away from an HMO. While not an HMO, there is an evolving concept which should be mentioned. Surgicenters of the free-standing type separate from the hospital such as the one at Phoenix, Arizona are of particular interest. These are of interest to the hospital because if such exists it will siphon off a percentage of those who would otherwise occupy a bed thereby reducing the daily census even further than it has dropped nationwide already. To the physician because it represents a 40 to 50 per cent reduction in cost to the patient in those cases where this "come-and-go surgery" can be done. It is of interest to note that the Joint Committee on Accreditation of Hospitals has underway (JCAH) consideration of methods for inspection and accreditation of these units and it goes without saying the third-party carriers are interested in this saving.

Involvement of physicians in any method of delivery of health care brings in review for the purpose of control not only of physicians but of all providers of health care, be they hospital, pharmacy or ambulances, and these reviews are not only of cost but of methodology and frequency of use as well as indications for therapy and any other kind of care. Physicians are developing and maturing Peer Review. Peer Review, as recently adopted by the House of Delegates of the AMA, is by definition the review of the practicing physician by the practicing physician and any other type of review is something else. In-hospital audit, tissue, utilization committee are all a form of review while claims review within the local insuring agent is review also.

In 1954, faced with demands of the longshoremen in Stockton, California for a pre-paid comprehensive plan for health services, the physicians there conceived and organized the San Joaquin Foundation for Medical Care which has remained active and healthy. Utilizing this as the model, the subject of Foundations is opened as another method of delivering health care and, while in existence for 17 years, must still be classified as experimental because they remain flexible in functions. Basically a Foundation for Medical Care is a corporation which has been organized separately from, but controlled by, the area Medical Society and its participating physicians. It functions to do utilization and claims review, to review practice patterns, to develop a Certified Hospital Admission Program (CHAP) (thereby reducing hospital and extended care facility stays) and can become interested in Comprehensive Health Planning, Multi-phasic Screening, Migrant Workers, and so on. The San Joaquin Foundation, which is the parent of the American Association of Foundations for Medical Care now numbering 35 groups across the country, has several basic points which must be enumerated.

1) The Foundation sets certain minimum standards of coverage which must be met before the Foundation will service that program for the insuring agent.

2) The Foundation takes over the processing of all claims as to eligibility, over and under utilization (both institutional and office), physicians' fees, hospital costs, and in many cases holds check writing authority from the insuring agent. Review is first by clerk matching against compiled norms and then by physician reviewer or to hospital reviewer or to the druggist reviewer. Since all data prints out together with the patient profile variants appear promptly (85 per cent of claims clear with no trouble) and the rest are viewed and either paid or mutually adjusted. None has ever gone to the Governing Board in 17 years for solution. Incidentally, reviewers other than the clerks are paid $25 per hour.

3) Known maximum allowances for physicians' fees are based on the California Relative Value Scale with a suitable conversion factor and are set by the Foundation as are all guidelines for all phases of care. These guidelines are reviewed and revised annually.

In Minnesota what was first known as the Hennepin County Foundation went statewide and received its initial financing as a joint venture of the Health Insurance Council and the Blue Cross-Blue Shield plans. It began operation in March 1971 and its status is not well publicized since it can not be judged for at least a year. It varies from the first one named (San Joaquin) in several ways: 1) Whereas San Joaquin insists on a minimum insurance standard in coverage sold before servicing a program, the Minnesota one has only suggestions to insuring agents but no requirements and will service all plans. 2) Guidelines of care and fees were developed by the Minnesota Foundation, relying heavily on the Ann Arbor studies of Payne, and these guidelines were furnished to the carriers who thereby retained their own clerical staffs and their own processing and payments mechanisms. Only those claims falling above or below the bell shaped curve are sent to the Foundation for review. 3) The maximum schedules for physicians' fees is not published as it was felt it might be inflationary, but the usual, customary and reasonable fee utilizing the 93rd percentile is the guide used.

The spread of the Foundation movement is of interest in the evolving Health Service Delivery System picture for a number of reasons: 1) It has been shown to effect about 11 per cent saving on the cost of care where instituted. 2) After start-up money the San Joaquin model has operated on a 3½–5 per cent of the premium charges. 3) It has been an educational and persuasive power to all providers of service as it shows exactly where they stand in relationship to the others. 4) Its efforts at CHAP need more evaluation. The theory is that in dollars it must cost less to move a patient along the chain of progressive care but the length of stay and the indications for variation must be carefully observed. 5) It affords a reliable and up-to-date method of Peer Review which is essential to the care being rendered.

In summary then, a number of ways the physician views the subject of the HMOs and other methods of developing health care plans have been reviewed. There are other variants which I will not mention except to name the Big Valley Project around Fresno, California concerned with Title 19 patients and the coordination effort developing at Cincinnati, Ohio involving all health agencies. In a pluralistic approach it is the firm conviction of the AMA and its Council on Medical Service that with the exception of the San Joaquin Foundation on Medical Care (which is a peculiar situation not applicable in the same form everywhere else), with this one exception, all others, including Kaiser-Permanante, must be studied as experimental and when their worth is proven as being of value to the people of this country they should be implemented in accordance with the principles of:

a) accessibility of care
b) quality of care
c) reasonable cost of care.

Surely all of us must not wish to dislocate abruptly present patterns until all persons in the health field are convinced the changes proposed will not do harm. The future of this soon to be the country's largest industry, the health field, needs a cooperative effort on the part of all its segments as together we try to reach our mutual goals—the welfare of the people of this country.

Prophylactic Considerations in Patients with Prosthetic Heart Valves Undergoing Dental Procedures

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Significant advancements have been made in understanding the pathogenesis and prophylaxis of endocarditis in congenital and acquired heart disease. In recent years, however, the internist cares for a great number of patients who have had prosthetic valve replacements. Prophylactic antibiotic consideration in these patients undergoing various dental procedures must be viewed in an entirely different light than those with naturally occurring valvular disease. Endocarditis in these patients, whether dating from the time of their dental procedure or occurring in the post-operative period have almost always proved to be fatal.[1] Several reports from the National Heart and Lung Institute[2,3] have described the clinical and pathological features of this complication. In each case of endocarditis involving the aortic prosthesis, the infection resulted in detachment of the prosthetic valve and massive fatal aortic regurgitation. The primary site of infection was found to be the tissues to which the prosthesis had been attached. The sutures had torn through the necrotic aortic annulus and the valve was partially or completely detached. In one of the patients, fatal endocarditis followed dental extractions, despite penicillin prophylaxis in doses similar to those recommended by the American Heart Association for patients with rheumatic heart disease.

These considerations suggest that a more comprehensive antibiotic regimen be given patients with prosthetic heart valves prior to dental manipulations to either control bacteremia or to prevent implantation and multiplication of the bacteria in the epithelium of the heart. One must emphasize that these manipulations include filling, cleaning, peridontal work as well as extractions. Unfortunately, bacteremia has been demonstrated to occur after such simple maneuvers as vigorous brushing of the teeth or even chewing hard candy. Multiple extractions or extensive procedures, particularly should be avoided in the presence of dental sepsis because of the increase in intensity of the bacteremia in this situation.

The following prophylactic antibiotic coverage is recommended prior to dental manipulation in patients with prosthetic valves. On the evening before the procedure give: 1) Penicillin V-500,000 units orally. One hour before the procedure give intramuscularly 2) Procaine Penicillin–600,000 units plus 3) Streptomycin–0.5 gm. plus 4) Crystalline Penicillin–600,000 units. Continue for three to five days following the procedure: 5) Procaine Penicillin–600,000 units I.M. every twelve hours plus streptomycin 0.5 gm. every twelve hours.

Alternatively, Penicillin V–500,000 units orally every six hours may be used in the post-operative period rather than the more desirable intramuscular procaine penicillin. If the patient is allergic to penicillin, equivalent doses of an alternate antibiotic (Erythromycin–0.2 gm. IM or 1.0 gm. orally or Keflin 0.5 gm. IM) should be given. If procedures are of necessity performed in an environment where penicillin-resistant staphylococci are likely contaminants, oxacillin 1–2 gms. orally every six hours may be added to the above program.

It is also important to point out that a complete dental evaluation with necessary treatment should be performed if possible prior to the operative replacement of heart valves. The previously recommended prophylactic antibiotic program of The American Heart Association[4] can be employed satisfactorily during these pre-operative manipulations.

In conclusion, recent advances in the operative treatment of heart disease carries a great deal of new responsibilities for the internist and cardiologist. These include many problems such an anticoagulant therapy, anesthesia for other operative procedures, intraoperative monitoring of the electrocardiogram etc. Adequate prophylaxis against endocarditis is but one of these considerations that may prevent an ensuing life threatening illness.

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Potential Legal Difficulties With Voluntary Sterilization

An unwed twenty-three year old woman comes to your office and requests a hysterectomy or a tubal ligation. She has recently obtained an abortion and seeks to avoid the chance of future pregnancy. She appears to be of sound physical and mental health and states that she has no future marital intentions. The hospital with which you are affiliated has an informal policy of performing voluntary sterilization only on married women who have obtained their spouse's consent and who, if they are under 30 years of age, have already given birth to two children. Will you be liable for a violation of the woman's constitutional rights if you refuse to perform the requested operation?

An irate husband upon whom you have recently performed a vasectomy comes to your office to announce his wife's recent pregnancy. What is your liability?

Although no Federal Court has announced that a woman has a constitutional right "to decide how many children she shall bear", *McCabe* v. *Nassau County Medical Center,* a recent decision by the United States Court of Appeals for the Second Circuit (essentially, New York) indicates that such a pronouncement may not be too far away. In that case, a woman went to a county medical center for the purpose of being sterilized, but was refused the operation on the basis of her failure to qualify under the hospital's age-parity formula. This formula established the number of children a woman of a particular age would have to have in order to qualify for sterilization. The woman sued the hospital for the violation of her right to decide for herself how many children she wanted. She claimed that the hospital's formula violated her right to privacy in her marital relationship and denied her equal protection of the law due to "irrational" distinction based on age. Her suit was brought both for injunctive relief, which would require the hospital to perform the operation, and for damages. The damages sought were $250,000 for "the constant state of fear of becoming pregnant" to which the woman was subjected during the interim, which allegedly caused great pain and increased the likelihood of further irreparable injury. The hospital subsequently performed the operation and moved to dismiss the case as being moot. The Court of Appeals held that the performance of the operation did not render the questions moot and that the lower court should have tried the issue of damages. The end result remains to be seen.

Despite the trend towards increasing liberality shown by society towards personal decisions of birth control, the only local jurisdiction which has a statute on the matter of voluntary sterilization is Virginia. At Chapter 27 of Title 32 of the Code of Virginia, express authorization is given to any physician or surgeon licensed by the state and acting in collaboration or consultation with at least one other physician to perform on any person 21 years of age or older a vasectomy or salpingectomy provided that a written request for the operation is made by such person 30 days prior to the performance of the operation and the consent of the spouse, if there is one, has been obtained. The statute further states that in the absence of negligence, no physician shall be liable either civilly or criminally by reason of having performed a vasectomy or salpingectomy as authorized by the statute.

This statute seems to serve the general purpose of dispelling any former doubts as to the legality of these types of operations, but leaves the performance of the operation to the discretion of the individual physician. The problem raised by the *McCabe* case is whether or not a patient has an enforceable constitutional right to become sterilized. If the lower court awards damages based on the violation of a constitutional right, sufficient precedent will be created for a future test case in the D.C. Metropolitan area.

If such a case is raised, what type of policies will be under attack? Evidently, certain hospitals in the area have rather strong policies against performing sexual sterilizations absent a true physiological need for the operation. A finding of a constitutional right to sterilization would preclude as a basis for a physician's decision to grant or refuse the operation any distinction between a physiological necessity and a purely voluntary decision to have the operation. Furthermore, any age-parity formula such as that referred to in the *McCabe* case would be unconstitutional, as would any distinction drawn between a married and a single woman.

A somewhat related issue is the liability of a physician when the spouse of a man who has had a vasectomy becomes pregnant. Although no local courts have decided on this matter, several other jurisdictions have had cases involving the liability of a physician based on the theory of a breach of contract to sterilize. In general, the contract to perform a surgical operation does not give rise to an implied "warranty to cure"; yet, the doctor and patient are free to go beyond this general rule by specifically including the end result of actual and effective sterilization as a term of their contract. Indeed, in cases of nontherapeutic sterilization the patient has requested the operation for a particular purpose and would normally want some form of assurance from the doctor that the procedure would accomplish actual sterilization. Hence, there is a potentiality of a doctor making an express agreement regarding the success of the operation. An agreement or assurance of this sort would constitute a form of warranty, the breach of which would render the doctor liable in a subsequent legal action.

In the cases involving vasectomy operations, the courts have recognized the possibility of a natural reversal of the operation (that is, a recanalization), and have allowed complaining patients to rely on this possibility to support their allegations that they have fathered a child. Furthermore, the courts may tend to take a dim view toward a doctor's defense based on the adultery of the wife. The drastic consequences of a finding of immoral conduct on the part of the wife and the subsequent illegitimation of the child could lead a court to adopt instead a position unfavorable to the doctor.

It would appear that the way to avoid liability for breach of contract (although not for negligence) is to assure a written understanding between the patient and the doctor before the operation is performed. Although all hospitals require sterilization patients to sign written consent forms authorizing the operation, the actual content of these forms varies. An effective form should contain an explicit acknowledgment by the patient that he understands the nature of the operation, that the success of the operation is not guaranteed and that he realizes the possibility of a natural reversal. Furthermore, the form should include explanatory terms in language that the layman could be expected to understand. Technical terms should be either fully explained or avoided.

The proper authorization forms would render a doctor innocent of any breach of contract, and the patient would have to base his allegations on negligence. In this case, the possibility of recanalization works to the doctor's advantage in that it allows the court to take notice of a hypothetical situation wherein a skillfully performed operation would not necessarily result in permanent sterility. This would make the patient's allegations of negligence increasingly more difficult to prove, as he would then have to present actual proof of negligence since the mere lack of success of the operation in and of itself would be insufficient to establish liability on the part of the physician.

A final word of caution. If the standard medical practice in the community with respect to vasectomies includes post-operative periodic sperm tests, the doctor might be found negligent, not for the operation itself, but for not conducting or advising the patient to obtain such tests. Therefore, to be on the safe side, a doctor should at least make the patient aware of the precautionary advisability of these periodic tests. Of course, the original consent form could be effectively utilized in this regard by containing the patient's acknowledgement that he is aware of the existence and advisability of these tests.

WILLIAM R. CHARYK

Edited by Stefan F. Tucker of Arent, Fox, Kintner, Plotkin and Kahn, Washington, D.C.

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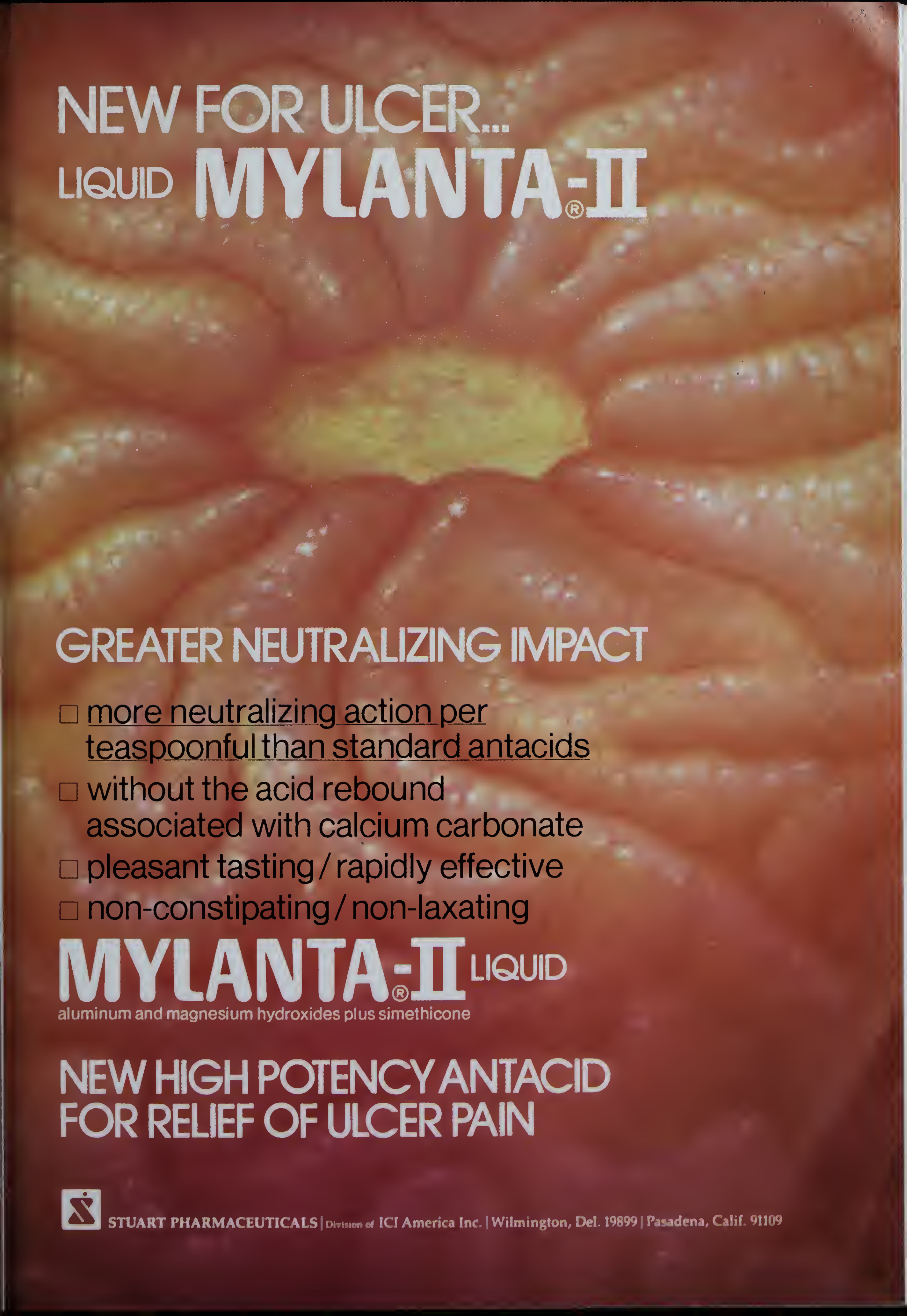

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Adverse Reactions: Nausea, headache, anorexia, vomiting, diarrhea, epigastric distress, abdominal cramping, constipation, a metallic, sharp and unpleasant taste, furry or sore tongue, glossitis and stomatitis possibly associated with a sudden overgrowth of *Monilia*, exacerbation of vaginal moniliasis, an occasional reversible moderate leukopenia, dizziness, vertigo, incoordination and ataxia, numbness or paresthesia of an extremity, fleeting joint pains, confusion, irritability, depression, insomnia, mild erythematous eruptions, "weakness," urticaria, flushing, dryness of the mouth, vagina or vulva, pruritus, dysuria, cystitis, a sense of pelvic pressure, dyspareunia, fever, polyuria, incontinence, decrease of libido, nasal congestion, proctitis, pyuria and darkened urine have occurred in patients receiving the drug. Patients receiving Flagyl may experience abdominal distress, nausea, vomiting or headache if alcoholic beverages are consumed. The taste of alcoholic beverages may also be modified. Flattening of the T wave may be seen in EKG tracings.

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For Trichomoniasis. In the Female: One 250-mg. tablet orally three times daily for ten days. Courses may be repeated if required in especially stubborn cases; in such patients an interval of four to six weeks between courses and total and differential leukocyte counts before, during, and after treatment are recommended. Vaginal Inserts of 500 mg. are available for use, particularly in stubborn cases. *When the vaginal inserts are used,* one 500-mg. insert is placed high in the vaginal vault each day for ten days and the oral dosage is reduced to two 250-mg. tablets daily during the ten-day course of treatment. Do not use the vaginal inserts as the sole form of therapy. *In the Male:* Prescribe Flagyl only when trichomonads are demonstrated in the urogenital tract, one 250-mg. tablet two times daily for ten days. Flagyl should be taken by both partners over the same ten-day period when it is prescribed for the male in conjunction with the treatment of his female partner.

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Warnings: **Do not use dietary potassium supplements or potassium salts unless hypokalemia develops or dietary potassium intake is markedly impaired.** Enteric-coated potassium salts may cause small bowel stenosis with or without ulceration. Hyperkalemia (>5.4 mEq/L) has been reported in 4% of patients under 60 years, in 12% of patients over 60 years, and in less than 8% of patients overall. Rarely, cases have been associated with cardiac irregularities. Accordingly, **check serum potassium during therapy, particularly in patients with suspected or confirmed renal insufficiency (e.g., elderly or diabetics). If hyperkalemia develops, substitute a thiazide alone. If spironolactone is used concomitantly with 'Dyazide', check serum potassium frequently** — both can cause potassium retention and sometimes hyperkalemia. Two deaths have been reported in patients on such combined therapy (in one, recommended dosage was exceeded; in the other, serum electrolytes were not properly monitored). Observe patients on 'Dyazide' regularly for possible blood dyscrasias, liver damage or other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving Dyrenium (triamterene, SK&F). Rarely, leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with the thiazides. Watch for signs of impending coma in acutely ill cirrhotics. Thiazides are reported to cross the placental barrier and appear in breast milk. This may result in fetal or neonatal hyperbilirubinemia, thrombocytopenia, altered carbohydrate metabolism and possibly other adverse reactions that have occurred in the adult. When used during pregnancy or in women who might bear children, weigh potential benefits against possible hazards to fetus.

Precautions: Do periodic serum electrolyte and BUN determinations. Do periodic hematologic studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in postsympathectomy patients. The following may occur: hyperuricemia and gout, reversible nitrogen retention, decreasing alkali reserve with possible metabolic acidosis, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), digitalis intoxication (in hypokalemia). Use cautiously in surgical patients. Concomitant use with antihypertensive agents may result in an additive hypotensive effect.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis; rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting (may indicate electrolyte imbalance), diarrhea, constipation, other gastrointestinal disturbances. Rarely, necrotizing vasculitis, paresthesias, icterus, pancreatitis, and xanthopsia have occurred with thiazides alone.

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Local Academic Training in Public Health

The administrative offices of the Community Health and Hospitals Administration, D. C. Department of Human Resources is a mini-campus where qualified employees may take training leading to a Master of Public Health degree. The degree program is a cooperative venture of the Department and the Johns Hopkins University School of Hygiene and Public Health, and is the only M.P.H. program in the metropolitan area where arrangements of this kind are made.

The degree program extends over a two-year period. Students participate in classroom activities part-time, thus enabling them to retain their status as full-time employees.

Eighteen students who are physicians, nurses, nutritionists, public health administrators, social workers or dentists, completed the first phase of the program in a five-week period during the summer of 1971. In this initial phase, full-time academic work was conducted in residence at the Johns Hopkins campus in Baltimore. Two subjects, Epidemiology and Biostatistics, were offered as introductory courses.

The second phase started in September 1971, and will continue through March 1973. For each quarter, enrollees take one subject and attend classes two or three half-days a week, depending upon course requirements. Some classes are offered on Saturdays, thus reducing the number of hours that students are away from the office or clinic. Courses taught in Washington, D. C. include Public Health Administration, Pathobiology (Biology of Populations), Maternal and Child Health and Population Dynamics, Environmental Health, Behavioral Sciences, History of Public Health, Medical Care and Hospitals, and Mental Health. Faculty commute from the Johns Hopkins University campus. The extension program is of equal high caliber as the curriculum given at the University in Baltimore.

The third phase, April through June 1973, is similar to the first one in that students will attend class at the Baltimore campus. However, this is negotiable and full-time attendance in Baltimore may not be required. Those who complete all requirements will receive a Master of Public Health degree in June 1973.

It is anticipated that the Metropolitan area campus curriculum will eliminate some of the health manpower problems facing the District of Columbia, improve the quality of health services, and provide opportunities for professional excellence.

Conducting the curriculum in an urban center has another advantage in that it offers opportunities for the University faculty to observe the practical problems which face the inner-city public health worker.

Started with a $20,000 grant from the D. C. Government, students are obliged to pay $2,700 tuition during the two-year period. This will increase to $3,000 in 1973 and it is anticipated that an additional increase in 1975 will bring the tuition to $3,300. Those in need of financial assistance make their own arrangements for loans and scholarships with the University or another source.

Facilities other than the administrative building of the Community Health and Hospitals Administration are used. Arrangements were made to use the laboratories of Howard University College of Medicine. Students are encouraged to use the facilities of the Library of Congress and the Library of Medicine. Other appropriate and available sites are utilized as needed.

Another class will start during July 1972, and will be open to non-governmental employees. The class will be limited to 25 degree candidates. Applicants must meet the entrance requirements of the University. These are:

1. graduation with a Bachelor of Science or other degree from a recognized college or university;
2. training and experience in medicine, dentistry, nursing, or one of the sciences with unusual experience or training in public health;
3. interest in scope of public health rather than a narrow field; and
4. recommendations from previous employers and educators.

The final selection of applicants is made by the University.

Applications are available from the Office of Training and Career Development, Community Health and Hospitals Administration, Room 418, 1875 Connecticut Avenue, Washington, D. C. 20009 or call (202) 387-6628.

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Elizabeth Morehead is an attractive receptionist at a downtown restaurant. She budgets her income, pays her own bills, and has time to serve as an assistant Girl Scout Leader two nights a week. She is, by any standard, a productive member of society. She used to be a resident at Forest Haven, the District's Institution for the mentally retarded.

Elizabeth was an unusual case, to be sure. Her level of intellectual functioning improved dramatically after a few months at Forest Haven. The interest shown to her by the staff, the emotional warmth of her companions in the new living quarters, the adequate diet, the enrichened environment of the modern classroom setting all combined to work the seeming miracle. The impoverished, nonaccepting environment she had come from had led Elizabeth into a private world within herself in which she no longer responded to environmental cues. Her growth rate declined physically as well as mentally. She was finally branded retarded by several indices.

Research has shown that retarded children can usually be helped to function at more complex levels if they are reached early enough in life with the right kinds of training and life experiences. The training can encompass almost any area of human functioning—at some level—and the life experiences can be any one of several kinds often found in American homes, provided that basic human acceptance of the child as a worthy individual in his own right is present. Another must is constancy or dependability of the emotional climate. The child must be able to predict events in his surroundings, especially those that seem to be generated by his own behavior. He must know in advance which of his behaviors will yield friendly responses from others around him and what behaviors will generate wrath. He must also be made aware of those emotional responses of persons around him that have nothing to do with any behavior of his, so that his growing self image will be based on reality.

Finally, if his brain is to attain its most complex level of functioning, the child must be provided with a wide diversity of afferent neuronal stimuli. Or in the language of the educator, he must be provided with an enrichened environment. Ralph, for example, can not tell the difference between coarse and fine sand paper when blindfolded. He has never stroked a fur-bearing animal, or run his fingers along the deep ridges in the bark of a pine tree. He is called a "leveler" where environmental stimuli are concerned. All perceptions are blended together in a bland admixture that lacks perceptual distinctions. He can't spot relevant details from among the irrelevant in any given situation, so he behaves in a manner that his friends derisively call stupid. Such derision increases his confused sense of emotional anguish and he retreats even farther from interactions with his environment and the derisive people in it.

This is one of the more fortunate histories of retardation, for Ralph, like Elizabeth, can be immensely helped at Forest Haven. Thanks to their expanding programs, he will one day be returned to the community as a productive citizen. His training and education will be thorough.

Apart from the more beneficial emotional climate that will be provided him, he will most likely enter Forest Haven's version of operation head start, in which he will be encouraged to interact with his environment in ever more complex ways. He will be sent on field trips to places where he has never been, to see sights, hear sounds, and smell odors that are new to him.

He will attend the ultra-modern school on the grounds of Forest Haven, where specially trained, highly qualified teachers will ferret out his special learning problems and give him individualized learning environments where necessary.

Meanwhile he will live, work and play with children who have various levels and kinds of handicaps, in order to further enrichen his environment and prepare him for the real world in which not everyone functions at the same complexity. Thus he is provided additional measuring posts of where he is psychologically, intellectually and physically: necessary ingredients in a sturdy, realistic self-concept that will provide him with emotional security for the trying days ahead when his snug environment at Forest Haven is replaced with ever more ambiguous environments outside the Institution.

First, he will be encouraged to take part in a sheltered workshop where he can learn a skill and begin to earn his own way financially. Then he will be encouraged to live with fellow workshop members in a less closely supervised house or apartment in town. Finally, he may obtain a job in the open job market and continue to live with selected peers under even less supervision.

Under this arrangement, he will budget his own funds, take care of his own clothing and transportation, and in some cases make his own arrangements for food. The supervisor will be on hand to answer any questions he might have concerning community resources, plus encourage him to undertake ever-broadening social encounters such as church activities, men's clubs or community recreation activities.

Other residents at Forest Haven who are not so fortunate as Ralph and Elizabeth, whose level of functioning can not be brought up to sufficient complexity to enable them to work in the community, will also be returned to the community. They, too, will take their place in as normal an environment as can be provided.

They will be placed with eight to ten others in homes such as houses or small apartment complexes within the community. They will have close supervision by two live-in houseparents in each unit. Otherwise their lives will be near normal. They will go on shopping excursions to neighborhood stores in small groups accompanied by one of the houseparents. They will even help shop for the groceries, have some decision picking out their clothing, and will help run their home in a democractic sort of way. They will attend neighborhood movies and recreational events in the evening and on weekends.

The supervisors will continue the resident training in self grooming and basic principles of social behavior. Their medical needs, as well as any other emergencies will be looked after by the resident staff.

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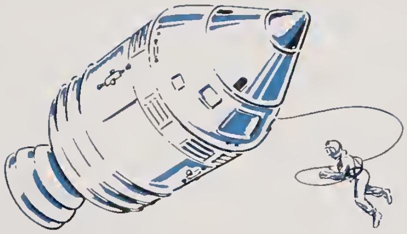

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Indications: Vaginitis due to Candida albicans and other Candida species.
Contraindications: Contraindicated for patients known to be sensitive to any of its components. During pregnancy manual Tablet or VAGELETTES Capsule insertion may be preferred since the use of the ointment applicator or tablet inserter may be contraindicated.
Caution: During treatment it is recommended that the patient refrain from sexual intercourse or the husband wear a condom to avoid re-infection.
Adverse Reaction: Clinical reports of sensitization or temporary irritation with CANDEPTIN Vaginal Ointment, Vaginal Tablets or VAGELETTES Vaginal Capsules have been extremely rare.
Dosage: One vaginal applicatorful of CANDEPTIN Ointment or one Vaginal Tablet or one VAGELETTES Vaginal Capsule is inserted high in the vagina twice a day, in the morning and at bedtime, for 14 days. Treatment may be repeated if symptoms persist or reappear.
Available Dosage Forms: CANDEPTIN Vaginal Ointment is supplied in 75 gm. tubes with applicator (14-day regimen requires 2 tubes). CANDEPTIN Vaginal Tablets are packaged in boxes of 28, in foil with inserter—enough for a full course of treatment. CANDEPTIN VAGELETTES Vaginal Capsules are packaged in boxes of 14 (14-day regimen requires 2 boxes.)

Store under refrigeration to insure full potency.

Federal law prohibits dispensing without prescription.

References: **1.** Olsen, J R Journal-Lancet *85* 287 (July) 1965. **2.** Giorlando, S W.: Ob/Gyn Dig. *13* 32 (Sept) 1971. **3.** Decker, A Case Reports on File, Medical Department, Julius Schmid **4.** Giorlando, S W., Torres, J.F., and Muscillo, G Am. J Obst. & Gynec. *90* 370 (Oct. 1) 1964 **5.** Lechevalier, H.: Antibiotics Annual 1959-1960. New York, Antibiotica Inc., 1960 pp 614-618 **6.** Friedel, H J Maryland M J , *15* 36 (Feb) 1966.

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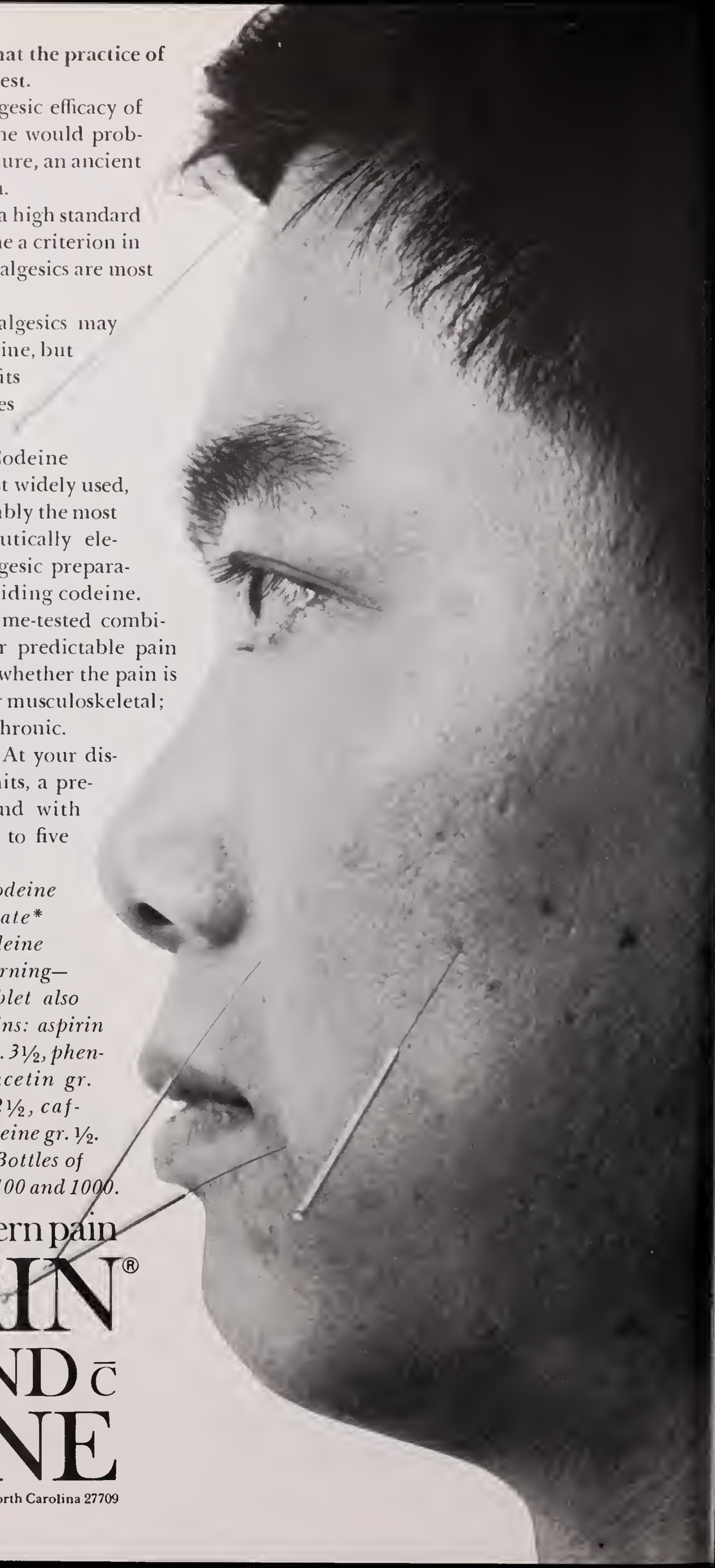

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The staff, meanwhile, will be continuously involved in a complete program of inservice training to upgrade their skills in understanding and working with the retarded. Staff training personnel will visit the living units and conduct on the spot training. Such training will be geared both to solving problems that the staff have encountered while working with the residents, and in achieving a more well-rounded, more homelike and community oriented life style for the residents.

As an aid to achieving a more normal atmosphere, the residents of a given unit will vary considerably in age, in level of handicap, and will contain both males and females. The only requirement for entering such homes will be that the resident be ambulatory, and not have a history of intractable antisocial behavior. There should be few of the latter at the end of the training programs at Forest Haven.

What will we need to accomplish this? Most problems, aside from low funding levels, are mechanical in nature. For example, we currently have three hundred residents ready to enter the community. At ten residents per living unit, that means 30 large houses or apartment complexes of various sizes. These are not available in the Metropolitan area.

As far as our environmental enrichment activities are concerned (mentioned above), we would like to have back the third of our vehicles that were recently removed from our in-house fleet, so the field trips can continue at a meaningful pace. And speaking of mechanical problems, we would like to have an ambulance to service our 1300 residents so that a resident with a broken leg will not have to sit up in the seat of the car all the way to the hospital.

All of our residents are not ambulatory, and never can be. There are special problems in working with these, to include many special arrangements that must be made. These will be the subject of next month's report.

ESSEX C. NOEL, III, M.D.
Director,
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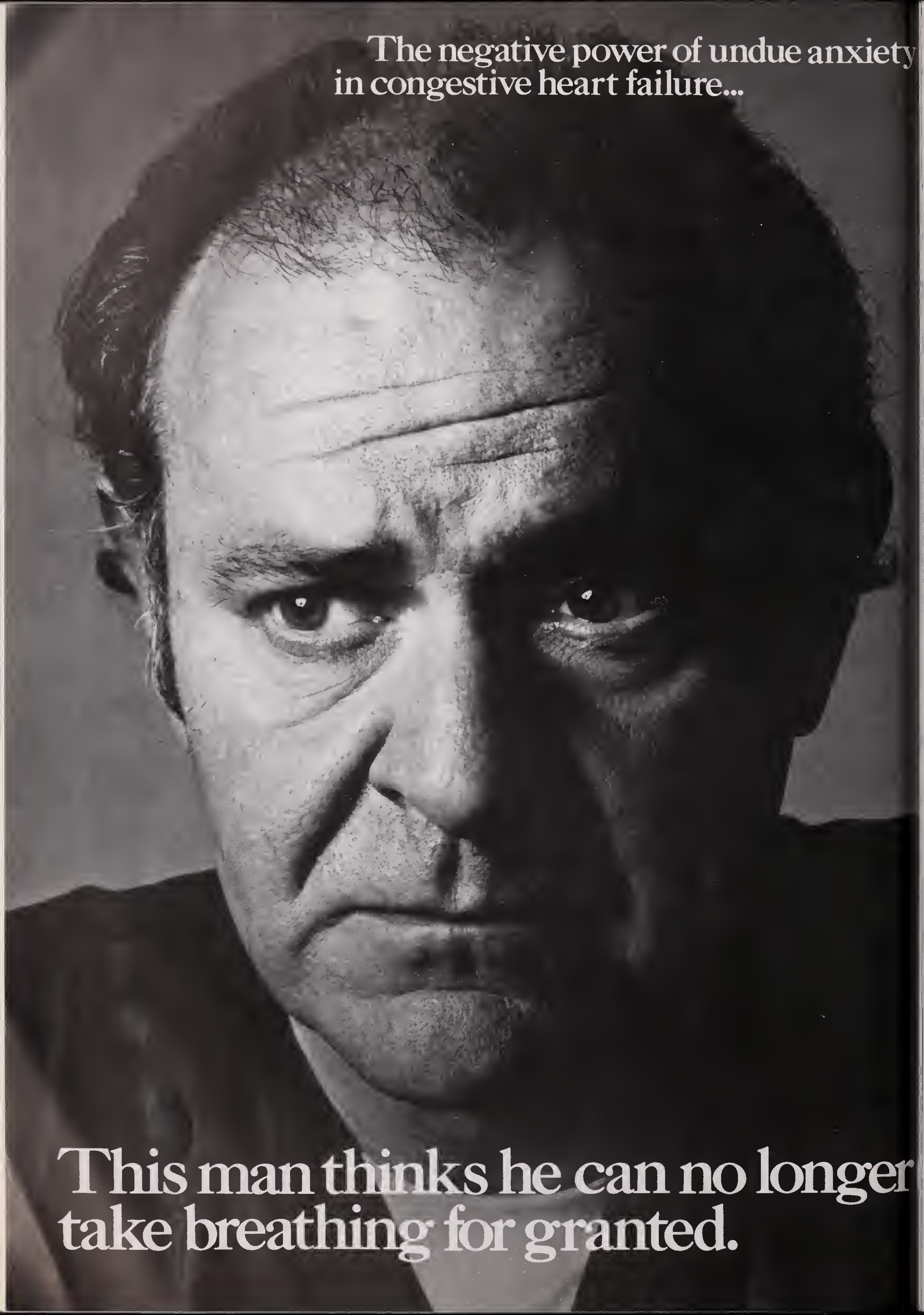
The negative power of undue anxiety
in congestive heart failure...
This man thinks he can no longer
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Typical of many patients with congestive heart failure, he also suffers from severe anxiety, a psychic factor that may influence the character and degree of his symptoms, such as dyspnea. His apprehension may also deprive him of the emotional calm so important in maintenance therapy.

Aid in rehabilitation

Specific medical and environmental measures are often enhanced by the antianxiety action of adjunctive Libritabs (chlordiazepoxide). Libritabs can also facilitate treatment of the tense convalescent patient until antianxiety therapy is no longer required. Whereas in geriatrics the *usual daily dosage* is 5 mg two to four times daily, the *initial dosage* in elderly and debilitated patients should be limited to 10 mg or less per day, adjusting as needed and tolerated.

Concomitant use with primary agents

Libritabs is used concomitantly with certain specific medications of other classes of drugs, such as cardiac glycosides, diuretics, antihypertensives, vasodilators and oral anticoagulants, whenever excessive anxiety or emotional tension adversely affects the clinical condition or response to therapy. Although clinical studies have not established a cause and effect relationship, physicians should be aware that variable effects on blood coagulation have been reported very rarely in patients receiving oral anticoagulants and chlordiazepoxide HCl.

The positive power of
Libritabs®
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5-mg, 10-mg, 25-mg tablets
t.i.d./q.i.d.
up to 100 mg daily
for severe anxiety accompanying congestive heart failure

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Indicated when anxiety, tension and apprehension are significant components of the clinical profile.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported. Use of any drug in pregnancy, lactation, or in women of childbearing age requires that its potential benefits be weighed against its possible hazards.

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Supplied: Tablets containing 5 mg, 10 mg or 25 mg chlordiazepoxide.

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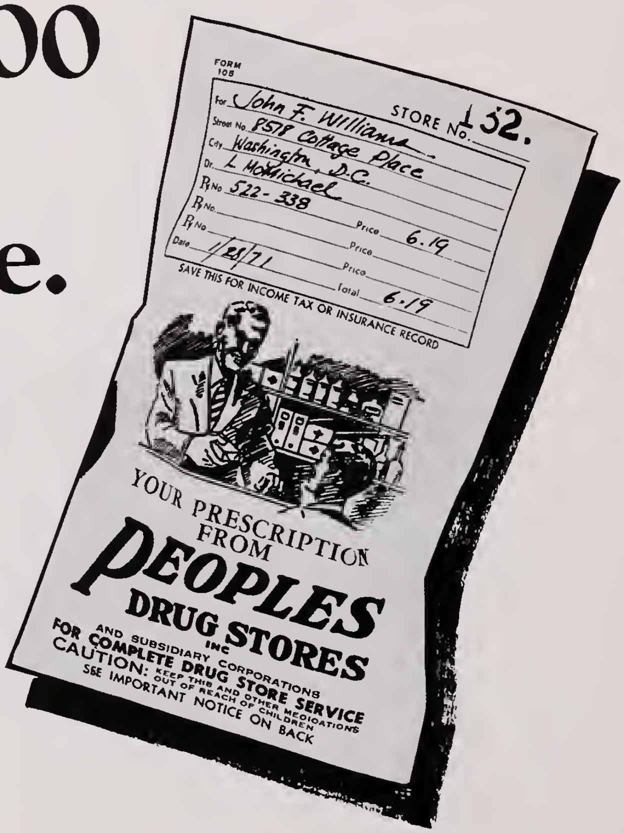

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DBI-TD® phenformin HCl
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Warnings: Use during pregnancy is to be avoided.
Precautions: 1. *Starvation Ketosis:* This must be differentiated from "insulin lack" ketosis and is characterized by ketonuria which, in spite of relatively normal blood and urine sugar, may result from excessive phenformin therapy, excessive insulin reduction, or insufficient carbohydrate intake. Adjust insulin dosage, lower phenformin dosage, or supply carbohydrates to alleviate this state. **Do not give insulin without first checking blood and urine sugar.**
2. *Lactic Acidosis:* This drug is not recommended in the presence of azotemia or in any clinical situation that predisposes to sustained hypotension that could lead to lactic acidosis. To differentiate lactic acidosis from ketoacidosis, periodic determinations of ketones in the blood and urine should be made in diabetics previously stabilized on phenformin, or phenformin and insulin, who have become unstable. If electrolyte imbalance is suspected, periodic determinations should also be made of electrolytes, pH, and the lactate-pyruvate ratio. The drug should be withdrawn and insulin, when required, and other corrective measures instituted immediately upon the appearance of any metabolic acidosis.
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Adverse Reactions: Principally gastrointestinal; unpleasant metallic taste, continuing to anorexia, nausea and, less frequently, vomiting and diarrhea. Reduce dosage at first sign of these symptoms. In case of vomiting, the drug should be immediately withdrawn. Although rare, urticaria has been reported, as have gastrointestinal symptoms such as anorexia, nausea and vomiting following excessive alcohol intake.
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—George Sarton, from "The History of Medicine Versus the History of Art"

Would it be useful in clinical practice to have government predetermine drugs of choice?

Opinion

Results of a survey of physicians:

13.3%
Yes, it would be useful.

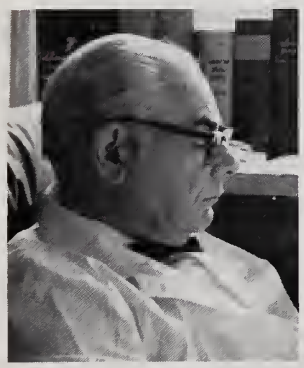
86.7%
No, it would not be useful.

Dialogue

Would it be useful in clinical practice to have government predetermine drugs of choice?

Doctor of Medicine

Walter Modell, M.D.,
Professor of Pharmacology,
Cornell University
Medical College,
Editor,
Clinical Pharmacology
& Therapeutics,
Drugs of Choice,
Rational Drug Therapy



The proposition that government should determine one or two "drugs of choice" within a given therapeutic class reflects the belief that a similarity in molecular structure insures a close similarity in pharmacologic effect. But this is by no means the rule. An obvious example would be in the field of diuretics, where a small change in chemical structure accounts for substantial differences in concomitant effects such as potassium excretion.

Any attempt to dictate the "drug of choice" would be complicated by the fact that some populations demonstrate a bimodal distribution in their reaction to drugs. If the data on drug response are mixed for the total population, one drug will appear to be as useful as the other. But if drug response is reported separately for different segments of the population, drug A will be found to be better for one group and drug B for the other.

It may, of course, be possible to determine drugs of choice in particular categories on a broad statistical basis. But there are always certain patients in whom a drug produces odd, unpredictable or idiosyncratic reactions. So, though a drug might statistically be the most useful one in a given situation, individual variations in response might make it the *incorrect* one.

The point I wish to make is that if two, three, four or more drugs in one class are of approximately equal merit, that in itself is justification for their availability. Exceptional cases do arise in which one drug would be useful to a certain segment of the population and another drug would be of no use at all. In the practice of medicine, the physician must be prepared to treat the routine as well as the unusual case.

Another objection to the determination of a drug of choice is that precise statements of *relative* efficacy are very difficult to make—much more difficult than statements of efficacy. For example, in testing drug efficacy, it is easy to determine the difference between a drug that is effective in treating a condition and one that is not at all effective. Thus, it is fairly easy to determine whether a drug is more effective than a placebo. But if you compare one drug that is effective with another drug that is also effective, and the relative differences between them are very slight, statements of relative efficacy may be very difficult to make with assurance.

I do not mean to imply that relative efficacy statements are not useful or can never be made. With some groups of drugs (e.g., analgesics), extensive study and precise methodology have yielded useful information on relative efficacy. But in most situations, such information can be acquired only through studies encompassing three to five years of use in many more patients than are used to compare drugs with a placebo for the introduction of a drug into commerce. It is really only after practitioners use a drug extensively that relative safety and efficacy in practice can really b determined.

The Bureau of Drugs ha suggested the package insert as a possible means o communicating informatio on relative efficacy of drug to the physician. I find thi objectionable, since I d not believe the physicia should have to rely on thi source for final scientifi truth. There is also a practical objection: Since fe physicians actually dispense drugs, they seldo see the package insert. I any event, I would maintain that the physicia should know what drug h wants and why without depending on the governmen or the manufacturer to te him.

Undoubtedly, physician are swamped by excessiv numbers of drugs in som therapeutic categories. An I am well aware that man drugs within such categories could be eliminate without any loss, or perhaps even some profit, t the practice of medicin But, in my opinion, neithe the FDA nor any othe single group has the expertise and the wisdom necessary to determine the or "drug of choice" in a areas of medical practice.

Maker of Medicine

neth G. Kohlstaedt, M.D.,
Vice President,
Medical Research,
li Lilly and Company

n my opinion, it is not
function of any govern-
t or private regulatory
ncy to designate a "drug
hoice." This determina-
should be made by the
sician after he has re-
ed full information on
properties of a drug,
then it will be based on
experience with this
g and his knowledge of
individual patient who
eking treatment.
an evaluation of com-
ative efficacy were to be
e, particularly by gov-
nent, at the time a new
g is being approved for
keting, it would be a
at disservice to medi-
and thus to the patient
e consumer. For exam-
when a new therapeu-
gent is introduced, on
basis of limited knowl-
, it may be considered
e more potent, more
ctive, or safer than
ducts already on the
ket. Conceivably, at
time the new drug
d be labeled "the drug
hoice." But as addi-
al clinical experience is
mulated, new evidence
become available.
er, it may be apparent
that the established products should not be so easily dismissed.

Variation in patient response to drugs constitutes one of the major obstacles to the determination of "drugs of choice." We are just beginning to open the door on pharmacogenetics, but it is evident that genetic differences cause wide variations in the way drugs are absorbed, metabolized, etc. This fact alone is sufficient to make unrealistic the idea that there is one drug in each class to be used for every human being.

The problem of determining relative drug efficacy is an extremely complicated one. Comparison with other drugs of the same class should not be a prerequisite for marketing a new substance. In some therapeutic areas, it may be difficult to make accurate comparisons. For example, in the treatment of infections it is not possible to conduct crossover studies. Recovery may be influenced by factors which cannot be controlled or measured, i.e., natural host resistance and virulence of infective agents. A drug's acceptability must often be judged on the basis of its own performance, and this may be limited to experience in a relatively small patient population. If the introduction of a new drug must await the adequate establishment of relative efficacy, the duration of clinical trial and extent of studies would be greatly prolonged, particularly for rare or unusual conditions. The availability of a new drug would be delayed. Many patients might suffer needlessly and lives might be lost.

Relative efficacy can best be established by experience in a general patient population through regular channels of clinical practice. The physician considers the patient as a whole, which means the patient often has multiple problems and drugs must be selected with this in mind. Hence, a "drug of choice" in an uncomplicated case may not be the best drug for a patient with associated problems. Publication of well-controlled studies in medical journals may provide comparative evidence; discussions at medical meetings, presentations at postgraduate courses, and the new audiovisual technology may bring evidence to physicians on comparative therapy. In a free medical marketplace, a drug that does not measure up will fall into disuse. For example, broad clinical experience has established vitamin B_{12} as the "drug of choice" for the treatment of primary pernicious anemia. No amount of advertising or promotional effort by the manufacturer could increase the use of liver extract for this anemia. However, a physician may wish to employ parenteral liver preparations for a special purpose.

In the field of surgery, peer review in the hospital has brought significant improvement in the use of new techniques and procedures. Something of this nature would be useful in the area of drug therapy. However, it should be developed by the medical profession itself and would necessitate, for its proper function, an improvement in the dissemination of reliable data on clinical pharmacology of drugs under consideration.

Ideally, information on the relative efficacy of drugs should be gathered and assessed by the physicians who actually administer the specific agents to a specific patient population. To do this, they will need even more information on the drugs they use – information that the pharmaceutical manufacturers must begin to provide if government regulation of *"drugs of choice"* is to be avoided.

Opinion & Dialogue

What is your opinion, doctor?
Send us your comments on the above issue.

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book REVIEWS

Immunological Disorders of the Nervous System. LEWIS P. ROWLAND, M.D., Editor. Price, $25.00. Pp. 470 with 81 illustrations. Baltimore: The Williams and Wilkins Company, 1971.

This volume represents the proceedings of the 47th Annual Meeting of the American Association for Research in Nervous and Mental Disease. Each year a selected panel of experts explores in depth a particular aspect of the nervous system. At various times, this may cover structure, mechanisms or functions of the nervous system, or diseases related to it. As in any such compilation of papers, there is not the even progression, step by step, that one finds in a volume prepared by one or several authors. Also, this volume presupposes a good deal of basic information on the subject of immunology. Many of the papers are technical, going into much detail which is important for the areas of research. However, there is a large amount of basic information which is applicable to clinical practice and will be of great interest to those involved in clinical medicine, not only in the field of neurology but also in general medicine. There are a number of papers outlining the immunopathology, the basic analyses of the immuno-proteins and a number of areas concerned with the animal models for the production of diseases which are similar to the so-called auto-immune diseases in the human. In addition, there is much discussion and detail regarding the immune globulin response both in the blood and spinal fluid. From a clinical point of view, there are specific papers which have excellent information. There is an important study of multiple sclerosis giving an excellent review of the current status of the immunologic aspects of this disease. The chapter on ideopathic polyneuritis is an up-to-date survey of the current knowledge, raising questions of interest to the research-minded and giving information for the clinician which is certainly of value.

The chapter and discussion of autoimmune disease is important and presents the current objections to the concept and raises questions as to its accuracy. The chapter on lupus erythematosus examines particularly the central nervous system manifestations. There is a short but important chapter describing, in brief, the central nervous system complications of organ transplantation.

Of historic interest for those who have been following the AARNMD proceedings is a followup paper by Gibbs and Gajdusek on the transmission of Kuru, Scrapie, Cretuzfelt-Jacob disease and mink encephalopathy which brings up-to-date the material presented at the 1964 meeting of the Association.

In general, this volume has followed the format of all previous volumes of this organization and certainly presents the most up-to-date details. Of great importance is the discussion at the end of selected chapters giving the current research activities, much of which has been unpublished to date.

Generally, this volume can be used with great advantage by those interested in research in the area of the immunology of the nervous system, not only the immunopathologic areas, but also in the biochemistry and in clinical research. It represents, in these areas, the most up-to-date research information much of which can be found in other literature but not so well summarized. On the clinical side the material is much less specific and the major value continues to remain in the area of neurology and internal medicine. There is very little information which would be applicable to the direct treatment and prognosis of patients with the various so-called "auto-immune" diseases but nonetheless there is useful information regarding diagnosis, particularly of the differential diagnoses of muscle disease, lupus and multiple sclerosis and polyneuritis.

While the material is over two years old, it is still timely. However, this raises the major criticism of the volume. Why does it take two years to edit such proceedings when the manuscripts are usually available immediately after the meeting?

DESMOND S. O'DOHERTY, M.D.

Hi, Sugar! VIVIAN G. LESSEL. Price, $4.95. Pp. 183. Tucson, Arizona: B & B Publishing Company, 1971.

Hi, Sugar! written by a diabetic who lived through thirty-six years of diabetes, presents facts and suggestions from her point of view in a rapid, easy to read form. The book is not intended to be a medical book nor a manual for self care, but a guide to diabetic "do's" and "don't" for successful diabetic living through the author's own experience of observation and factual information.

While reading *Hi, Sugar!*, one is immediately impressed with the author's enthusiasm for giving encouragement and confidence to the new diabetic and brightens the patient's outlook towards his problems and tried to help him and his family to over-come the fears and insecurity of the disease.

Even though there have been fine books written for the diabetic, HI, SUGAR! should be considered as a helpful and interesting introduction to the subject from the lay point of view and can be recommended to the diabetic and his family.

WILLIAM KURSTIN, M.D.

The Psychiatric Interview in Clinical Practice. ROGER A. MACKINNON, M.D. AND ROBERT MICHELS, M.D. Price, $12.50. Pp. 473. Philadelphia: W. B. Saunders, 1971.

Interviewing is the most important tool of the mental health professional, and interviewing skill is a result of one's personality, the clinical experiences of oneself and others, and the concepts of mental illness that one uses. This book describes experiences and concepts that many will find useful additions to their own.

The authors' focus is somewhat narrow. They see the interview as a systematic attempt to understand the relationship of psychopathology to emotional conflicts. This orientation does not see determining the need for medications as part of the interviewing process with schizophrenics, and it does not see determining the usefulness of behavioral therapy as part of the interview process with phobic patients. Their goal is to help the interviewer get past the initial phase of individual psychoanalytically oriented psychotherapy.

The core chapters consist of a description of a major patient grouping, e.g., depressed patients, schizophrenic patients, etc., followed by psychoanalytic concepts that apply to that patient group, and then the management of the interview with that type of patient is spelled out. The descriptions are clear and what few technical terms are used are already known to most beginning psychiatric residents. In interviewing, one needs concepts that are simple and explicit enough to direct one's actions from moment to moment, and the authors meet that requirement. Situations and issues that will be met in interviewing a diagnostic grouping are pictured, and overall strategy and specific tactics to meet the situations are not hedged, but specific. Much of the wealth of the material probably arises from the authors' experiences since the references that are listed are far too inadequate to be the source. For example, the section on hysterical personality is as full of useful therapeutic maneuvers as one will find in a single writing on that topic, yet at least five of the thirteen references for that chapter say nothing useful about hysterical personalities.

In addition to the core chapters on interviewing various types of patients, there are sections on general interviewing principles, general psychoanalytic concepts, crisis interviewing, ward patient consultation, handling telephone calls, and ways of recording the interview.

Overall, this book ranks as one of the best on interviewing and most mental health trainees will gain considerably in their management of patients through absorbing the contents of this book.

ROGER PEELE, M.D.
Director, Area D Community Mental Health Center Saint Elizabeths Hospital and Clinical Associate Professor of Psychiatry and Behavioral Science, George Washington University

Growth Control in Cell Cultures. G.E.W. WOLSTENHOLME AND JULIE KNIGHT, Editors. Price, $7.20. Pp. 275 with illustrations. Edinburgh: Churchill Livingstone, 1971.

"Growth Control in Cell Cultures" is a collection of thirteen highly technical papers presented as a Ciba Foundation Symposium on factors relating to cell growth.

The papers deal, among other things, with the inhibition of growth created by crowded cellular conditions, contact inhibition, sensitive cell surface sites, the contributions of serum components to growth regulation and of other environmental factors such as local PH.

The papers are highly technical and obviously prepared for the sophisticated virologist and those knowledgeable in the field of oncogenic viruses and cell culture. Most physicians, even those devoted to oncological pursuits, would find these papers difficult to follow, tiresome to digest and definitely not material for a moment of pleasant relaxation

PHILIP H. PHILBIN, M.D.

Changing Concepts in Cardiovascular Disease. HENRY I. RUSSEK, M.D. AND BURTON L. ZOHMAN, M.D. Price, $23.75. Pp. 475 with illustrations. Baltimore: The Williams & Wilkins Company, 1972.

Another new book in cardiology? Yes, exactly my initial sentiments, but this one was delightful reading and well worth the time it took to do so. Despite the contents having been based upon edited proceedings of "The American College of Cardiology by St. Barnabas Hospital Symposium," of December, 1970, the contents are quite appropriate for 1972. The 37 chapters written by different contributors account for some variability in style and readability but in almost all instances, were concise and comprehensive. There is a certain amount of overlap in discussion of topics such as that on "Physiologic basis of anti-anginal therapy" and "Intractable angina pectoris" but it is not to any significant objectionable degree. For those who have kept up with all the current literature in the cardiovascular field—the book will offer nothing new. However, those who have been unable to keep up with everything will find this compilation rather informative, and an efficient way of being updated. The reader will find exciting and useful information that deal with advances in areas such as myocardial ultrastructure, myocardial oxygen consumption, electrophysiology, cardiac drug pharmacology, and the clinical management of major cardiac problems. There is a little of everything for those interested in basic information, clinical and bedside management, and surgical advances in the management of both congenital, acquired valvular and coronary artery disease. Adequate references after each chapter enables the reader to pursue his interest in a particular topic in greater detail. This compilation should appeal most of all to the physician in practice with an active interest in cardiovascular diseases.

Of special interest are the chapters which deal with the advances in physiology and therapy of ischemic heart disease, congestive heart failure, cardiac arrhythmias, and the chapters that deal with stress testing and physical fitness. An important and provocative chapter is devoted to diseases that result from the many interventions that a physician undertakes in the current management of his patients for which a new word "periontogenic diseases" has been coined. The book most certainly lives up to its title and is in itself a course in postgraduate education. An excellent addition to the library of anyone involved in cardiovascular diseases.

ANTONIO C. DELEON, JR., M.D.

The Education of a Doctor: My First Year on the Wards. JOHN MACNAB, M.D. Price, $6.95. Pp. 222. New York: Simon & Schuster, 1971.

John MacNab, M.D., in his book, *The Education of a Doctor, My First Year on the Wards,* tells us in a humorous and praiseworthy vein that medical education is a gradual process by which the student passes naturally into clinical situations while becoming more and more proficient with the knowledge and language of medicine.

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In commendable style, the author transforms these experiences into light and enjoyable reading. His accounts, in diary form, develop like mystery stories until the apprentice finally assumes the role of the physician. He stresses the obvious importance of the preclinical sciences to clinical practice. When the embryo physician first confronts the mysteries of the hospital ward, he must become familiar with the technical arts of medicine, learn how to work as a member of a medical team, and acquire skill and sensitivity in dealing with people as patients. The degree to which the student of medicine masters these challenges determines how smoothly he fills the clinical role.

It is a pleasure to recommend this volume to all students of medicine.

EDISON MIYAWAKI, M.S.

Psoriasis: Proceedings of the International Symposium. Stanford University, 1971. EUGENE M. FARBER, M.D. AND ALVIN J. COX, M.D., Editors. Price, $18.50. Pp. 478 with illustrations. Stanford, California: Stanford University Press, 1971.

A four-day symposium on psoriasis was held at Stanford University in 1971. Many investigators from all of the major continents participated in this meeting, and the fifty-one presentations that were delivered there comprise the book *Psoriasis*, edited by Eugene M. Farmer, M.D. and Alvin J. Cox, M.D.

The three major sections of this volume pertain to clinical manifestations, basic biological phenomena, and therapy of psoriasis. Many of the chapters represent original studies and there are some new ideas in this text. No major breakthroughs are to be found to provide answers to the conundrums of psoriasis, a disease that is thought to affect between 2 and 8 million Americans.

Approximately one-fifth of this book is devoted to methotrexate treatment of psoriasis and its possible relationship to hepatic damage. No firm conclusions are reached in these chapters on methotrexate therapy, but the general tenor is that of caution in the use of antimetabolites for psoriasis and the desirability of careful patient selection when such therapy is used. Very careful monitoring of hepatic function is strongly encouraged in patients receiving methotrexate for psoriasis.

This book will be useful to serious students of basic biological aspects of psoriasis. Those chapters devoted to clinical manifestations and therapy, while useful to the practitioner of dermatology, are not superior to material that can be found in basic dermatology texts and selected journal articles.

JAMES P. SHAPER, M.D.

Lasers in Medicine. LEON GOLDMAN, M.D. AND R. JAMES ROCKWELL, JR. Price, $35.00. Pp. 385 with illustrations. New York: Gordon and Breach Science Publishers, Inc., 1971.

This book reviews the extensive and detailed work of the staff of the Laser Laboratory of the University of Cincinnati, the only laboratory of its kind which was specifically established for the actual use of laser radiation in all areas of medicine and biology including both basic research and clinical investigative studies.

The first six chapters introducing the characteristics of laser radiation are apparently written by Mr. Rockwell, Directing Physicist of the Laboratory. These chapters are excellently written, and, although obviously directed toward a medical audience, still require a sound background in physics and electronics for clear understanding of the material.

The following eighteen chapters are directly related to the medical application of the laser, including tissue reaction, holography, animal investigations, safety, ophthalmology, dermatology, cancer research and surgery, and specialized applications. Dr. Goldman, a dermatologist, would appear to have covered the general areas well in his reviews of his own and work from other laboratories. This reviewer, familiar with the studies in ophthalmology, but not in the other disciplines, was favorably impressed with the organization, detail, proper emphasis, and recent studies covered in the review.

For the physician seriously interested in particular applications of the laser, and certainly for libraries where lasers are presently available or becoming available, the book is recommended.

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obituaries

William Ronald Strong, M.D.
1921–1972

Dr. William R. Strong, a general surgeon known for his work as a burn specialist, died May 13, 1972. He resided at 7208 Loch Lomond Drive, Bethesda, Maryland.

He was born in West Point, New York and received his undergraduate education at the Citadel at Charleston, South Carolina. He received his M.D. degree from the George Washington University here and served his internship and residency at Emergency Hospital, now the Washington Hospital Center.

A resident physician at the Bethesda Naval Medical Center while in the Navy, Dr. Strong served as an orthopedic surgeon and later was officer in charge of the U.S. Naval Tissue Bank. Responsible for the design and modification of freeze-drying equipment used for processing human transplants, he continued to serve as a consultant to the Naval Medical School on tissue preservation.

A member of the Washington Board of Police and Fire Surgeons, he was well known here as a burn specialist and for his frequent appearances at fires. He also worked with the Police department's Civil Disturbance Unit and received a special award "in appreciation for his interest, knowledge, and perseverance in the creation of the first Intensive Care Burn Unit to serve the citizens of the Washington Metropolitan community."

As a member of the Medical Society of the District of Columbia, Dr. Strong served actively on the Emergency Medical Service Committee and was its Vice Chairman from 1963 to 1970. He was also a member of the American Medical Association, the American Burn Association and the Southern Medical Association.

He is survived by his wife, Valda Berzins Strong, of the home.

James Edwin Wissler, M.D.
1906–1972

Dr. James E. Wissler, a Washington area radiologist and former senior partner of Drs. Groover, Christie and Merritt, died May 15, 1972. He resided at 8620 Fenway Drive, Bethesda.

Born in Waynesboro, Virginia, he grew up in Manassas. He received his M.D. degree from the University of Virginia and served his internship and residency at the University of Virginia Hospital.

Dr. Wissler came to Washington in 1934 and joined the radiology group of Drs. Groover, Christie and Merritt.

In addition to his membership in the Medical Society of the District of Columbia, Dr. Wissler was a member of the American Medical Society and the Southern Medical Association. He was also a fellow of the American College of Radiology.

He is survived by his wife, Lola Johnson Wissler, of the home address; a daughter, Mrs. Roger W. Stallkamp, of Dayton, Ohio, and four grandchildren.

Howard Irving Weinstein, M.D.
1912–1972

Dr. Howard I. Weinstein, associate director for medical affairs in the bureau of product safety of the Food and Drug Administration, died April 23, 1972. He resided at 2445 Lyttonsville Road, Silver Spring, Maryland.

Born in New York, New York, he received his B.S. degree from the College of the City of New York and his M.D. degree from the University of Vienna. He served his internship and residency at the Beth Israel Hospital in New York City.

He served in the Pacific as a lieutenant colonel in the Army Medical Corps during World War II and received the Purple Heart.

Dr. Weinstein had been a physician with the Government for 25 years and had been an Active member of the Medical Society of the District of Columbia since 1947.

He is survived by his wife, Anna, of the home; two daughters, Mrs. Deborah Ruth Ingel, of Austin, Texas, and Marsha Joan, of Bethesda; his mother, Kate, of Silver Spring; a sister, June, of Arlington, and a brother, Lawrence, of New York.

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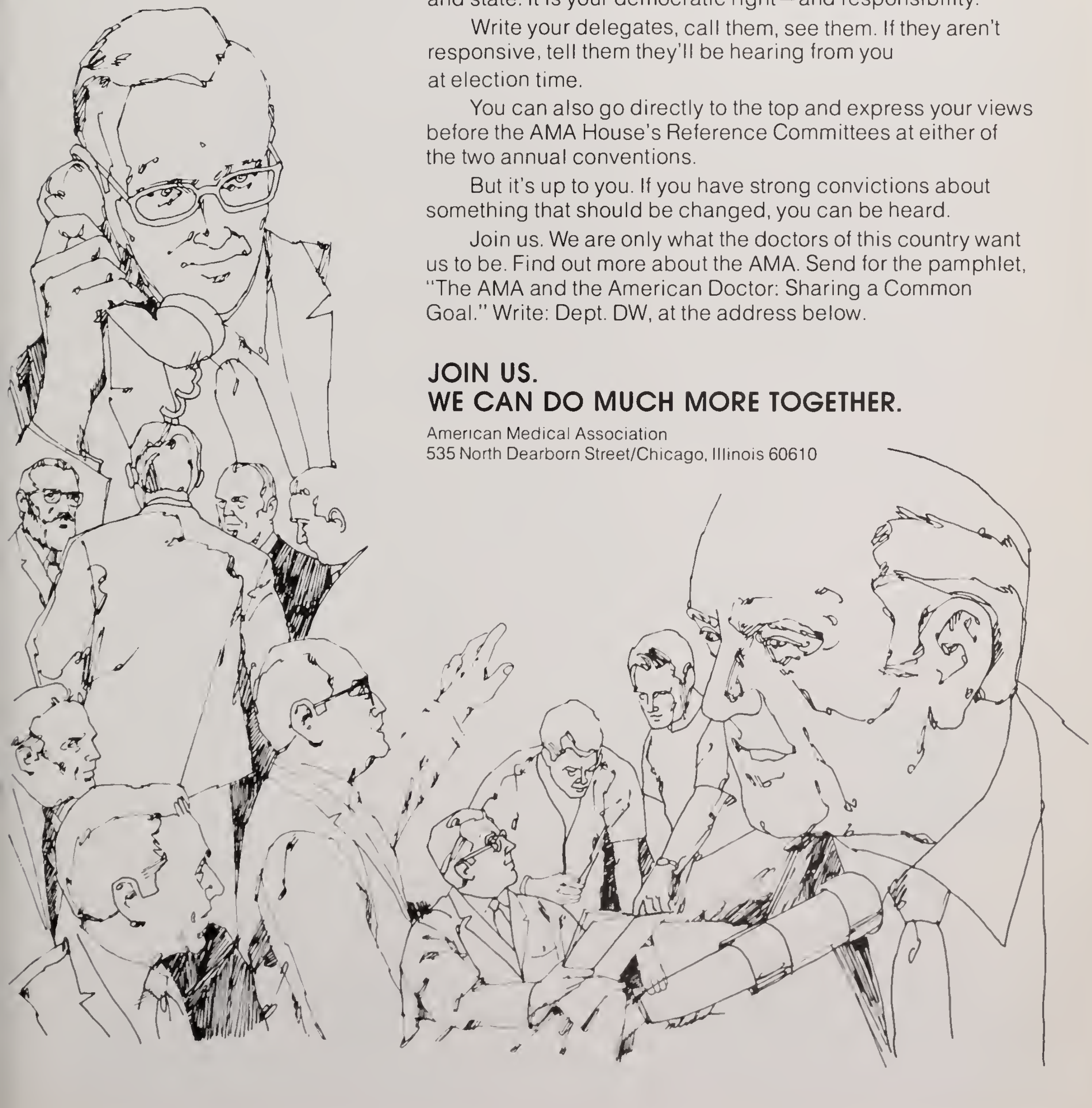

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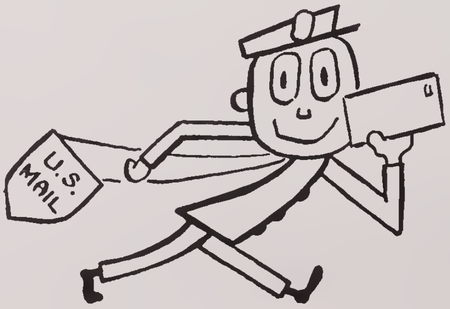

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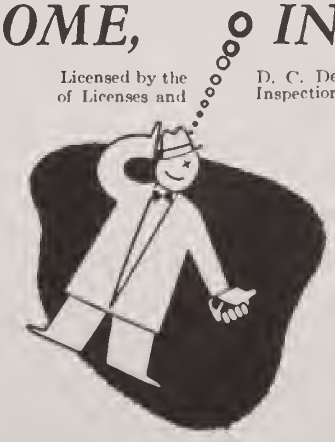

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Should children be deprived of milk because of "lactose intolerance?"

No, according to the Protein Advisory Group of the United Nations.

In a special report, the PAG stated, "It would be highly inappropriate, on the basis of present evidence, to discourage programs to improve milk supplies and increase milk consumption among children because of fear of milk intolerance." The statement emphasizes that low lactase activity and results obtained in tests with high lactose loads are not adequate indications of milk intolerance.

The statement also affirms the advisability of using milk as an excellent source of protein in child feeding programs.

Dairy Council is sponsoring further research on lactose intolerance. This is just another of the many areas of interest of Dairy Council, in the pursuit of better health for everyone through sound nutrition practices.

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Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence. In pregnancy, lactation or women of childbearing age, weigh potential benefit against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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PRESIDENT'S MESSAGE

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FIRST IN WAR...

FIRST IN PEACE...

FIRST IN THE PERFORMANCE OF ILLEGAL (?) ABORTIONS

A rather dubious distinction, but one which our Capital City seems destined to achieve. With five out-patient abortion facilities now operating and others reported in the planning stage, abortion on demand is an every-minute reality here in Washington. Exact figures are not available, but by checking abortion facility statements, 1,000 weekly abortions appears a conservative estimate. A substantial number of these are from out of the city, and "planeloads" of 20 or 30 candidates arriving from distant cities in this country and Canada is not uncommon.

Yet the legality of the procedure is still far from certain. Indeed, unless "necessary for the preservation of the mother's life or health," the act is still prohibited. The Supreme Court has upheld this statute, but has ruled that if an infraction of the statute is suspected, the burden of proof is on the prosecution to plead and prove that the abortion was not necessary as defined. Since this would be extremely difficult to accomplish, no test case has been initiated, and those operating abortion facilities seem confident it will not be.

Because of this rather fine point of law, our City Council had felt it unwise to issue regulations governing the operations of abortion clinics: to do so might imply approval of an illegal operation. Yet numerous instances of questionable practices in regard to these clinics were called to the attention of officers of our Society. An official policy as regards abortion was adopted by the Executive Board on February 22, 1971, as follows:

> "That when termination of pregnancy is in conformity with the laws of the District of Columbia, the interruption of a pregnancy should be a matter between a licensed doctor of medicine and his patient, and that the doctor will be governed by the same prevailing professional standards and ethics of medical practice which govern all medical-

rrell C. Crain, M.D.

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> surgical procedures. It is understood that appropriate consultation should be obtained when indicated."

It therefore seemed wise to ask the Committee on Maternal Welfare to cooperate with the Section on Obstetrics and Gynecology in drawing up a set of guidelines which would define good medical practice as it applies to abortions performed in a facility not connected with a hospital. Open to question is whether many abortions now being performed are necessary "for the preservation of the mother's life or health." Furthermore, many of our members hold serious moral reservations regarding the ethical practice of these abortions. Nevertheless, as physicians of the Medical Society of the District of Columbia, we can neither ignore nor disregard our responsibility to the citizens of this city to insist that since abortions are being performed in the city without hindrance from the legal authorities who are aware of their performance, these must be done in accordance with good medical practice. The Executive Board has therefore authorized the publication of appropriate guidelines as soon as they are completed by the Committee on Maternal Welfare and the Section on Obstetrics and Gynecology.

The City Council, too, has now decided to move forward and enact regulations for the licensing and control of out-patient abortion facilities. Both sets of regulations prohibit the performance of abortions in a physician's office unless expanded to include administrative, counseling and laboratory personnel. As these steps are accomplished, our city will have strong weapons for dealing with this difficult problem.

Darrell C. Crain

SAINT ELIZABETHS HOSPITAL 1855-1972

"A prophet," the Bible tells us, "is not without honour save in his own country, and in his own house." Saint Matthew's devastating observation about prophets applies with equal precision to Saint Elizabeths Hospital, a venerable institution whose scientific work is reported in this issue of the ANNALS. Known for many years throughout the world as a leader in hospital psychiatry and scientific research, Saint Elizabeths has not escaped criticism (both deserved and undeserved) from sources closer to home. Described by friends as a "model psychiatric hospital with splendid research and treatment facilities" and by foes as "a snakepit of a state hospital for the District of Columbia," one is bewildered by the huge discrepancy in points of view. The truth, one suspects, lies somewhere in the middle with an administration clamoring for more support and an insufficient staff struggling to cope valiantly with the deluge of enormously difficult problems deposited at its doorsteps. It is profoundly hoped that Dr. Luther Robinson, the new Superintendent, will be given sufficient help to fulfill the objectives of the hospital's illustrious founder, Dorothea Lynde Dix, to provide "the most humane care and enlightened curative treatment of the insane of ... the District of Columbia."

In 1955, on the occasion of the centennial celebration of the founding of Saint Elizabeths, I observed, "For 100 years Saint Elizabeths Hospital has stood on the imposing heights above the confluence of the Anacostia and Potomac Rivers, commanding an unparalleled view of the Capitol of the United States." In a curious way, this beautiful location of the hospital may have contributed to its sense of "splendid isolation," for it is sad but true that the hospital, until quite recently, did precious little to involve itself with the mental health needs of the community it served. For too many years it acted merely as a depository for the grossly aberrant. There were no facilities for follow-up care of discharged patients or outpatient clinics in the city staffed by Saint Elizabeths personnel. The recent development of an active community Mental Health Center under the leadership of Dr. Roger Peele shows great promise and is definitely a step in the right direction.

Obviously much more needs to be done and the new leadership will need all the help it can get. Saint Elizabeths still operates under the National Institute of Mental Health which in turn is responsible to the Department of Health, Education and Welfare. There has been much talk in recent years of transferring the hospital from the Federal Government to the District of Columbia. It would be catastrophic if such a transfer were to occur before the hospital and the District are really ready for it. This may take years but the time is certainly not now.

Saint Elizabeths has a proud record of distinguished scientific and humanitarian achievement. It now needs to redouble its efforts to become an integral part of the community it serves so that one may eventually be able to say truthfully that Saint Elizabeths Hospital and the District of Columbia are separated only by the Anacostia River.

Zigmond M. Lebensohn, M.D.

STATEMENT CONCERNING FROZEN RED CELL CONCENTRATES

The recent availability of frozen red cell concentrates through the Blood Program of the Washington Regional Red Cross Blood Center has prompted the following definition of the clinical indications for this specialized component.

Red cell concentrates frozen at -80°C in glycerol are prepared for transfusion by 1) thawing at 37°C, 2) continuous flow washing to remove the cryoprotective agent (glycerol) and 3) resuspension in 0.8% sodium chloride and 0.2% glucose, buffered to pH 7.4.

Since frozen red cell concentrates are washed and resuspended in an electrolyte solution, the hazards associated with whole blood transfusions - citrate toxicity, hyperkalemia, post-transfusion hepatitis and irregular antibodies - are minimized or eliminated. The risk of immunologic sensitization to leukocyte, platelet and plasma antigens is also reduced.

The initial indication for frozen red cell concentrates was the transfusion of patients with rare blood cell types or red cell antibodies for whom appropriate blood was not otherwise readily available. More recent experience has demonstrated the usefulness of frozen red cells in additional clinical situations.

Organ transplantation candidates who require transfusions may receive frozen red cell concentrates to minimize the risk of sensitization to transplantation (HL-A) antigens which are present on leukocytes and platelets. Persons sensitized to non-red cell antigens by pregnancies or previous transfusions and who have a history of recurrent non-hemolytic transfusion reactions usually tolerate frozen red cell concentrates because of the absence of leukocytes and platelets in the washed preparations. Similarly, persons who are massively or frequently transfused, such as during chronic renal dialysis, open heart surgery or other major surgical procedures, may benefit from the decreased risk of hepatitis sensitization to non-red cell antigens.

Physicians within the District of Columbia requiring frozen red cell concentrates should make appropriate arrangements through the Director of their hospital Blood Bank. Specimens of recipient blood will be cross-matched against aliquots of the frozen red cell concentrates at the Washington Regional Red Cross Blood Center. If compatible, appropriate red cell concentrates will be thawed, deglycerolized and prepared for transfusion. Under the most favorable circumstances, the total elapsed time from receipt of the specimen to availability of the washed red cells at the Washington Regional Red Cross Blood Center is approximately 1-1/2 hours. Frozen red cell concentrates have a 24-hour outdating from time of deglycerolization because of entry into the sterile container during the washing process. The current hospital participation fee for each 250 cc red cell concentrate is $50.

In summary, transfusions of frozen red cell concentrates are now a practical reality. Although the cost may seem high, the cost benefits achieved through the reduction in post-transfusion hepatitis, minimal transplantation antigen sensitization, and inventory economy should offset the direct costs of the specialized processing.

Subcommittee on Frozen Blood of the
Blood and Tissue Committee
Medical Society of D.C.

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unaltered by the drug. Use with caution in first trimester of pregnancy and in nursing mothers. Drug may appear in cord blood and breast milk. Serious, even fatal, blood dyscrasias, including aplastic anemia, may occur suddenly despite regular hemograms, and may become manifest days or weeks after cessation of drug. Any significant change in total white count, relative decrease in granulocytes, appearance of immature forms, or fall in hematocrit should signal immediate cessation of therapy and complete hematologic investigation. Unexplained bleeding involving CNS, adrenals, and G.I. tract has occurred. The drug may potentiate action of insulin, sulfonylurea, and sulfonamide-type agents. Carefully observe patients taking these agents. Nontoxic and toxic goiters and myxedema have been reported (the drug reduces iodine uptake by the thyroid). Blurred vision can be a significant toxic symptom worthy of a complete ophthalmological examination. Swelling of ankles or face in patients under sixty may be prevented by reducing dosage. If edema occurs in patients over sixty, discontinue drug.

Precautions: The following should be accomplished at regular intervals: Careful detailed history for disease being treated and detection of earliest signs of adverse reactions; complete physical examination including check of patient's weight; complete weekly (especially for the aging) or an every two week blood check; pertinent laboratory studies. Caution patients about participating in activity requiring alertness and coordination, as driving a car, etc. Cases of leukemia have been reported in patients with a history of short- and long-term therapy. The majority of these patients were over forty. Remember that arthritic-type pains can be the presenting symptom of leukemia.

Adverse Reactions: This is a potent drug; its misuse can lead to serious results. Review detailed information before beginning therapy. Ulcerative esophagitis, acute and reactivated gastric and duodenal ulcer with perforation and hemorrhage, ulceration and perforation of large bowel, occult G.I. bleeding with anemia, gastritis, epigastric pain, hematemesis, dyspepsia, nausea, vomiting and diarrhea, abdominal distention, agranulocytosis, aplastic anemia, hemolytic anemia, anemia due to blood loss including occult G.I. bleeding, thrombocytopenia, pancytopenia, leukemia, leukopenia, bone marrow depression, sodium and chloride retention, water retention and edema, plasma dilution, respiratory alkalosis, metabolic acidosis, fatal and nonfatal hepatitis (cholestasis may or may not be prominent), petechiae, purpura without thrombocytopenia, toxic pruritus, erythema nodosum, erythema multiforme, Stevens-Johnson syndrome, Lyell's syndrome (toxic necrotizing epidermolysis), exfoliative dermatitis, serum sickness, hypersensitivity angiitis (polyarteritis), anaphylactic shock, urticaria, arthralgia, fever, rashes (all allergic reactions require prompt and permanent withdrawal of the drug), proteinuria, hematuria, oliguria, anuria, renal failure with azotemia, glomerulonephritis, acute tubular necrosis, nephrotic syndrome, bilateral renal cortical necrosis, renal stones, ureteral obstruction with uric acid crystals due to uricosuric action of drug, impaired renal function, cardiac decompensation, hypertension, pericarditis, diffuse interstitial myocarditis with muscle necrosis, perivascular granulomata, aggravation of temporal arteritis in patients with polymyalgia rheumatica, optic neuritis, blurred vision, retinal hemorrhage, toxic amblyopia, retinal detachment, hearing loss, hyperglycemia, thyroid hyperplasia, toxic goiter association of hyperthyroidism and hypothyroidism (causal relationship not established), agitation, confusional states, lethargy; CNS reactions associated with overdosage, including convulsions, euphoria, psychosis, depression, headaches, hallucinations, giddiness, vertigo, coma, hyperventilation, insomnia; ulcerative stomatitis, salivary gland enlargement.
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>
> *—George Sarton, from "The History of Medicine Versus the History of Art"*

Would it be useful in clinical practice to have government predetermine drugs of choice?

Opinion

Results of a survey of physicians:

13.3%
Yes, it would be useful.

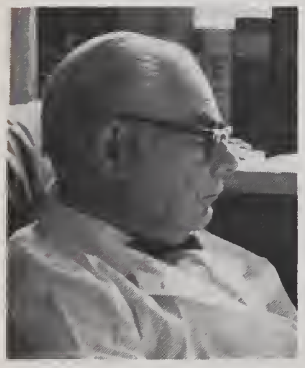
86.7%
No, it would not be useful.

Dialogue

Would it be useful in clinical practice to have government predetermine drugs of choice?

Doctor of Medicine

Walter Modell, M.D.,
Professor of Pharmacology,
Cornell University
Medical College,
Editor,
Clinical Pharmacology
& Therapeutics,
Drugs of Choice,
Rational Drug Therapy



The proposition that government should determine one or two "drugs of choice" within a given therapeutic class reflects the belief that a similarity in molecular structure insures a close similarity in pharmacologic effect. But this is by no means the rule. An obvious example would be in the field of diuretics, where a small change in chemical structure accounts for substantial differences in concomitant effects such as potassium excretion.

Any attempt to dictate the "drug of choice" would be complicated by the fact that some populations demonstrate a bimodal distribution in their reaction to drugs. If the data on drug response are mixed for the total population, one drug will appear to be as useful as the other. But if drug response is reported separately for different segments of the population, drug A will be found to be better for one group and drug B for the other.

It may, of course, be possible to determine drugs of choice in particular categories on a broad statistical basis. But there are always certain patients in whom a drug produces odd, unpredictable or idiosyncratic reactions. So, though a drug might statistically be the most useful one in a given situation, individual variations in response might make it the *incorrect* one.

The point I wish to make is that if two, three, four or more drugs in one class are of approximately equal merit, that in itself is justification for their availability. Exceptional cases do arise in which one drug would be useful to a certain segment of the population and another drug would be of no use at all. **In** the practice of medicine, the physician must be prepared to treat the routine as well as the unusual case.

Another objection to the determination of a drug of choice is that precise statements of *relative* efficacy are very difficult to make—much more difficult than statements of efficacy. For example, in testing drug efficacy, it is easy to determine the difference between a drug that is effective in treating a condition and one that is not at all effective. Thus, it is fairly easy to determine whether a drug is more effective than a placebo. But if you compare one drug that is effective with another drug that is also effective, and the relative differences between them are very slight, statements of relative efficacy may be very difficult to make with assurance.

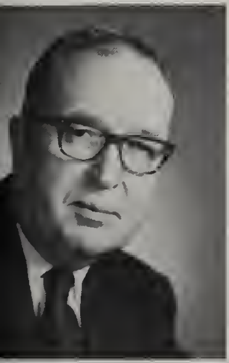
I do not mean to imply that relative efficacy statements are not useful or can never be made. With some groups of drugs (e.g., analgesics), extensive study and precise methodology have yielded useful information on relative efficacy. But in most situations, such information can be acquired only through studies encompassing three to five years of use in many more patients than are used to compare drugs with a placebo for the introduction of a drug into commerce. It is really only after practitioners use a drug extensively that relative safety and efficacy in practice can really determined.

The Bureau of Drugs h suggested the package i sert as a possible means communicating informati on relative efficacy of dru to the physician. I find th objectionable, since I not believe the physici should have to rely on th source for final scienti truth. There is also a pra tical objection: Since f physicians actually di pense drugs, they seld see the package insert. any event, I would mai tain that the physici should know what drug wants and why without pending on the governme or the manufacturer to t him.

Undoubtedly, physicia are swamped by excessi numbers of drugs in so therapeutic categories. A I am well aware that ma drugs within such ca gories could be eliminat without any loss, or p haps even some profit, the practice of medic But, in my opinion, neit the FDA nor any ot single group has the exp tise and the wisdom nec sary to determine the "drug of choice" in areas of medical practice

laker of Medicine

eth G. Kohlstaedt, M.D.,
Vice President,
ledical Research,
Lilly and Company



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that the established products should not be so easily dismissed.

Variation in patient response to drugs constitutes one of the major obstacles to the determination of "drugs of choice." We are just beginning to open the door on pharmacogenetics, but it is evident that genetic differences cause wide variations in the way drugs are absorbed, metabolized, etc. This fact alone is sufficient to make unrealistic the idea that there is one drug in each class to be used for every human being.

The problem of determining relative drug efficacy is an extremely complicated one. Comparison with other drugs of the same class should not be a prerequisite for marketing a new substance. In some therapeutic areas, it may be difficult to make accurate comparisons. For example, in the treatment of infections it is not possible to conduct crossover studies. Recovery may be influenced by factors which cannot be controlled or measured, i.e., natural host resistance and virulence of infective agents. A drug's acceptability must often be judged on the basis of its own performance, and this may be limited to experience in a relatively small patient population. If the introduction of a new drug must await the adequate establishment of relative efficacy, the duration of clinical trial and extent of studies would be greatly prolonged, particularly for rare or unusual conditions. The availability of a new drug would be delayed. Many patients might suffer needlessly and lives might be lost.

Relative efficacy can best be established by experience in a general patient population through regular channels of clinical practice. The physician considers the patient as a whole, which means the patient often has multiple problems and drugs must be selected with this in mind. Hence, a "drug of choice" in an uncomplicated case may not be the best drug for a patient with associated problems. Publication of well-controlled studies in medical journals may provide comparative evidence; discussions at medical meetings, presentations at postgraduate courses, and the new audio-visual technology may bring evidence to physicians on comparative therapy. In a free medical marketplace, a drug that does not measure up will fall into disuse. For example, broad clinical experience has established vitamin B_{12} as the "drug of choice" for the treatment of primary pernicious anemia. No amount of advertising or promotional effort by the manufacturer could increase the use of liver extract for this anemia. However, a physician may wish to employ parenteral liver preparations for a special purpose.

In the field of surgery, peer review in the hospital has brought significant improvement in the use of new techniques and procedures. Something of this nature would be useful in the area of drug therapy. However, it should be developed by the medical profession itself and would necessitate, for its proper function, an improvement in the dissemination of reliable data on clinical pharmacology of drugs under consideration.

Ideally, information on the relative efficacy of drugs should be gathered and assessed by the physicians who actually administer the specific agents to a specific patient population. To do this, they will need even more information on the drugs they use – information that the pharmaceutical manufacturers must begin to provide if government regulation of *"drugs of choice"* is to be avoided.

Opinion & Dialogue

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Saint Elizabeths Hospital

LUTHER D. ROBINSON, M.D.*

We are grateful to the Medical Society of the District of Columbia for devoting this issue of *Medical Annals of the District of Columbia* to Saint Elizabeths Hospital. As a means of sharing professional and scientific knowledge with the reading public in the field of psychiatry and mental health, contributions are continually being made to the literature by members of the Medical Society of Saint Elizabeths Hospital.

Two years have passed since a progress report has been made on the activities of the hospital. During that period many changes have taken place. Primary among the changes was the deactivation of the National Center for Mental Health Services, Training, and Research last year. Upon the deactivation of the National Center, Saint Elizabeths Hospital created the Overholser Training and Research Division to carry out training and research activities. The Division is carrying out the mission of its predecessors and has made many innovations. Its newly created newsletter, "Trends in Training," gives an account of its very worthwhile activities.

I am happy to state that the evacuation of the 19th century dilapidated, uninhabitable buildings was completed. The consolidation of Forensic Programs into one division is now complete. A drug-free therapeutic community-type rehabilitation program for drug addicts of Health Service Area D of the District of Columbia was created in a halfway house on campus as a part of the Area D Community Mental Health Center's program. Also for Area D citizens, a program for adolescents was developed. A new program for children and adolescents from other areas of the District of Columbia was created. A new admission service has been established in the former geriatric division allowing the age-integration of patients. Under a Hospital Improvement Program Grant a behavior modification program was established in Que Division. The Mental Health Program for the Deaf moved into its own physical facility with a full-time staff. Our number of Board-Certified psychiatrists nearly tripled from 5 to 14. The number of deaths dropped by almost 100, from 485 in fiscal year 1970 to 394 in fiscal year 1971. The average number of inpatients dropped by 910, from 4,075 in April 1970 to 3,165 in April 1972. The increase in the average number of outpatients for the same period was 508, up from 2,092 to 2,600. Also, the number of patients on the end of the month rolls decreased by 513, from 6,278 to 5,765. We are now carrying out educational and training programs in conjunction with Federal City College. One is the Washington Opportunities for Women's program to train men and women from the disadvantaged community to become social-work aides. The other is the Department of Health, Education and Welfare's Upward Mobility College to provide educational opportunities for lower-paid employees to prepare them for additional skills, facilitate their advancement, and to improve services. We have a greater degree of citizen participation, particularly in our Area D Community Mental Health Center.

Saint Elizabeths Hospital is a 3,400 bed Federal mental health facility occupying a beautiful 320-acre campus on a promontory in Southeast Washington, D. C., offering an imposing view of the nation's capital and environs. Organizationally it is located in the National Institute of Mental Health. The hospital admits approximately 4,000 patients annually under provisions of the District of Columbia Hospitalization of the Mentally Ill Act, Federal and District of Columbia Codes governing criminal proceedings, and other Federal and District of Columbia Codes. It serves primarily residents of the District of Columbia but also certain other categories of patients. Its average patient load is approximately 5,900. Of these, approximately 3,350 are treated as inpatients in 26 of the hospital's 103 buildings. Approximately 2,550 patients are treated on an outpatient basis. Accordingly, the hospital treats approximately 10,000 patients annually. The patient load represents the full spectrum of medical-psychiatric disorders and socio-economic levels. Psychoneurotic and personality disorders are encountered with increasing frequency as we move closer toward community-based mental health care.

The hospital's mission is three-fold, namely, treatment (including care and rehabilitation), training, and research. To carry out its mission, the staff

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Based on a paper presented at the 34th Annual Meeting of the Medical Society of Saint Elizabeths Hospital, April 14, 1972.

Reprint requests may be addressed to Dr. Robinson at Saint Elizabeths Hospital, Washington, D.C. 20032.

consists of approximately 3,800 employees in areas of direct patient care, administration, and support services. Volunteers contribute invaluable services to the hospital's programs. The staff uses an eclectic approach to accomplish its mission, drawing upon all available treatment modalities, mental-health facilities, and personnel, while at the same time through research and training tries to find and develop new resources.

General therapeutic and rehabilitative activities include but are not limited to individual and group psychotherapy, family therapy, psychodrama, socio-therapeutic group meetings, counseling, occupational therapy (industrial therapy and educational rehabilitation), education, recreational therapy (art, drama, music, sports, games, etc.), dance therapy, behavior modification, psychiatric nursing, medication, electroshock therapy, hydrotherapy, milieu therapy, vocational rehabilitation, and religious ministry.

The hospital is divided into 10 clinical psychiatric divisions. Three of these provide services for patients referred from community mental health centers of three of the Health Service Areas of the District of Columbia. A fourth division operates a comprehensive community mental health center for Health Service Area D. Three others serve no-fixed-address residents and nonresidents of the District of Columbia. The three remaining divisions have special programs–one for forensic psychiatry, one for deaf patients, and one for support to research activities of other programs of the National Institute for Mental Health. Moreover, the hospital has special treatment and rehabilitation programs for children and adolescents, alcoholics, and drug-dependent patients. Beyond this, a full range of medical, surgical, and laboratory services are available to patients. Saint Elizabeths provides inpatient, outpatient, and partial hospitalization services, emergency services 24 hours per day, consultation and education, precare and aftercare, training, research, and evaluation.

The hospital is accredited by the Joint Commission on Accreditation of Hospitals. Its training programs are approved by appropriate accrediting bodies such as the American Medical Association, the American Psychological Association, the National League for Nursing, and others.

As we look toward the future we are in the process of developing an organized system of unitization which will give the hospital's divisions a greater degree of autonomy in providing services to patients through the team concept. This in turn will allow continuity of care and closer contacts with patients and the community in the overall rendering of quality care in areas of prevention, treatment, and rehabilitation. Thus, the hospital recognizes a state of interdependence with the community as it carries out its role in the national mental health effort.

On behalf of Saint Elizabeths Hospital I express deep appreciation to the employees and others whose dedicated efforts have contributed to the hospital's progress. We look forward to a very bright future.

Mental Health Training and Research Programs at Saint Elizabeths Hospital

DANIEL D. COWELL, M.D.*

Emerging from the deactivation of the National Center for Mental Health Services, Training, and Research in June, 1971, the Winfred Overholser Training and Research Division of Saint Elizabeths Hospital is nearing completion of its first year of educational and research activities.

During this period the new Overholser organization has consolidated the activities of 50 staff employees and 80 trainees from 13 different training programs in carrying on the functions of both the former Vestermark Division of Intramural Training and the former Overholser Division of Clinical Research. The new Division is making a major effort to provide the highest quality professional training and research programs in accordance with its mandate as the principal training and research arm of Saint Elizabeths Hospital.

Historically, each of the Hospital's training programs had its own sense of development, its own traditions and policies, and each had helped to make Saint Elizabeths a rich training base of great diversity. There nevertheless has been a need to provide for central coordination of these programs with respect to interests which they share, for the satisfaction of mutual concerns requiring administrative, financial and consultative support, and for the pooling of resources to permit assembly of special multidisciplinary educational programs and projects.

To meet these needs the Overholser Division was formed and accorded responsibility for conducting accredited rotating internship and psychiatric residency programs, as well as an internship in Dentistry and residency training in Ophthalmology and Pathology. Graduate programs are also carried out in those non-medical disciplines related to the mental health field such as Clinical Psychology, Psychodrama, Nursing, Recreational and Occupational Therapy, Social Service, Dance Therapy, and the Catholic and Protestant Chaplaincies. The Division additionally provides and supervises affiliations, clerkships, and practical experience in Medicine, Social Service, Nursing, Clinical Psychology, the Rehabilitation Therapies, and Volunteer Services. It fosters cooperative relationships with institutions of higher learning, such as the George Washington University and its Medical School, and is available for collaboration with programs of the Division of Manpower and Training of the National Institute of Mental Health in areas of mutual interest.

Additional dimensions of the Overholser training mission include collaboration with other Saint Elizabeths Hospital components in providing extensive in-service training in psychiatric nursing and in developing numerous training programs in group dynamics and group therapy. Special educational clinics and programs in mental health are conducted by the Division for community clergymen and for law-enforcement agencies such as the Metropolitan Police Department and the U. S. Secret Service.

The Research component of the Division investigates psychological, ecological and cultural aspects of normal, criminal and assaultive behavior, shares responsibility for the technical review and evaluation of research conducted throughout the Hospital, fosters cooperative relationships with educational institutions in areas which concern research and teaching, and participates upon request in evaluating the effectiveness of training and treatment programs.

Five different types of medical student programs, including a clinical clerkship in Psychiatry for George Washington University medical students, complement the 30 paid positions currently filled by rotating interns and psychiatric residents. The Overholser Division's nine-man Psychiatry Training Faculty which conducts these programs is supplemented by a large staff of supervisors and senior consultants with the didactic program of seminars, demonstrations and rounds emphasizing an interdisciplinary, community-oriented approach to clinical services.

Among the Non-Medical Training Programs, the Group Training Section conducts eight major interdisciplinary Hospital group-work programs, while Clinical Psychology provides a varied clinical or research experience for 15 psychology graduate students. The Protestant Chaplaincy offers a full-time, three-year mental-health training program for clergy, placing heavy emphasis upon clinical community assignments in Mental Health Service Area D in Southeast Washington, while the Catholic Chaplaincy provides a one-year training program in clinical pastoral work for priests. Psychodrama, Social Service, Occupational Therapy, Recreational Therapy and Dance Therapy sections provide substantial service to patients in addition to offering paid training positions for undergraduate and graduate matriculants. Nursing Education supports mental health training for several hundred students per quarter from ten nursing schools at the Diploma, Bachelor, Associate and Master's Degree levels.

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Educational activities, newly developed this year, have included a Training Newsletter, a Spring Family Therapy Symposium, an Overholser Day of clinical presentations by trainees and invited guests, and vigorous support for the Annual Meeting of the Medical Society of Saint Elizabeths Hospital. In this connection, I am pleased to note the contributions to this issue of the *Medical Annals of the District of Columbia* which have been made by trainees and staff of the Overholser Division.

The Division is grateful for having been presented with this opportunity of delineating its programs for the readers of the *Medical Annals.*

THE VIEW BOX

Prepared by
The Department of Radiology
Washington Hospital Center

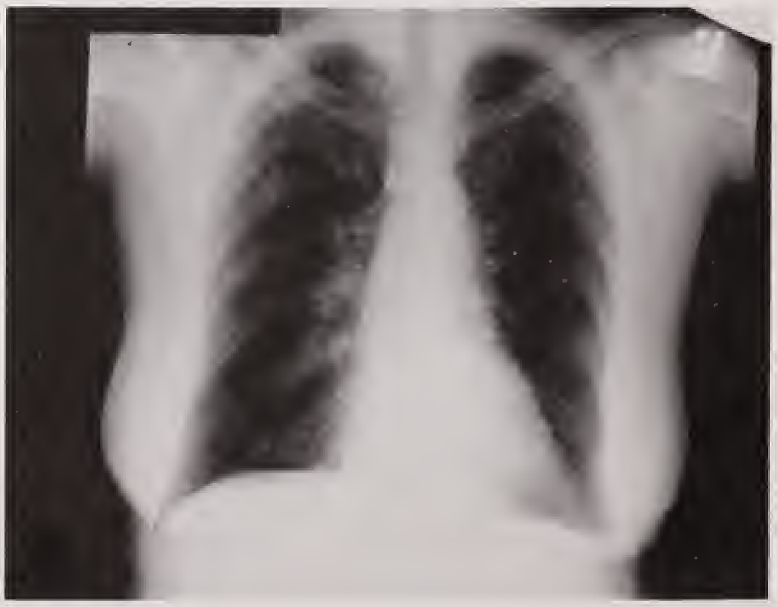

FIG. 1. PA Chest

A 78 year old white female presented with complaints of recurrent gastrointestinal bleeding. Physical examination was unremarkable. Barium Enemas and Upper Gastrointestinal series were consistently negative. Arteriography was not performed. Laboratory data was essentially negative. Radiographs of the chest revealed an irregular nodular density in the right upper lobe.

(For diagnosis see page 530)

Organizing to Improve Services: The Structuring of Public Mental Health Services

ROGER PEELE, M.D.*

During the past decade, there have been trends in the organization of public mental health services that parallel developments in large organizations of other medical specialities such as Medical Society Foundations and Health Maintenance Organizations. The same trend is also seen in efforts to establish local "one stop" human service centers. These trends represent an effort to improve the responsiveness of the services. This paper will discuss the structuring of one of the largest and most experienced of medical organizations: the public mental health services.

The *raison d'etre* of public mental health services is the assumption of responsibility for the mental health of people regardless of ability to pay, regardless of the timing of illness, and regardless of the type or severity of the illness. This includes being responsible for the various types of prevention. (The degree to which this *raison d'etre* is not met is usually the degree to which one should ask why have the public services at all and not just rely on the private sector.) Thus, providing structure for public mental health services should be met in terms of how responsibility is determined and how the organization decides who does what to whom.

This paper will describe the functional components that form mental health organizations and how they can be used in proposing a structure for public mental health services. The very important topic of the actual planning process itself will not be covered. Instead, the focus will be on suggestions that a health professional can bring to the planning process. Additionally, this paper will be limited to the structure of clinical activities, not budgeting activities, clerical activities, etc.

In looking over the structures of State mental health departments, city mental health agencies, public hospitals, and community mental health centers, one finds six ways of organizing clinical responsibilities:

1. *By time of entry.* Responsibility is determined by when the client entered the mental health delivery system. Examples include a mental health professional or unit being given responsibility for each admission in order, being assigned all admissions coming in during the mornings, or being assigned cases admitted on Wednesdays.

2. *By mode of delivery.* Responsibility is determined by what modality of service the client needs. Examples include maximum security unit, inpatient ward, day-care unit, outpatient unit, consultation program, public education unit, social action unit, and family therapy unit.

3. *By profession.* Responsibility is determined by which professional group can best treat the client's needs. Examples include psychology department, pastoral counseling services, nursing services, social work services, mental health counselor services, medical services, and psychodrama department.

4. *By psychiatric problem* ("categorical"). Responsibility is determined by the patient's psychiatric problem or diagnosis. Examples are drug-abuse programs, alcoholic programs, chronic-care programs, acute-care programs, and hyperactive child-service care.

5. *By patient characteristics.* Responsibility is determined by a non-pathological characteristic such as sex, race, age, and language. Examples are children's wards, Spanish-speaking units, and program for the deaf.

6. *By population.* The responsibility is determined by a community, an industry, or an institution to which the individual belongs. Examples are community mental health centers, union mental health programs, and mental health services for military personnel.

Nearly any large public mental health service has the first five models, and during the past decade there has been a marked increase in the use of the by-population model. See Table 1 for a simplified organization of the Area D Community Mental Health

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Based on a paper presented at the 34th Annual Meeting of the Medical Society of Saint Elizabeths Hospital, April 14, 1972, entitled "Structuring Public Mental Health Services."

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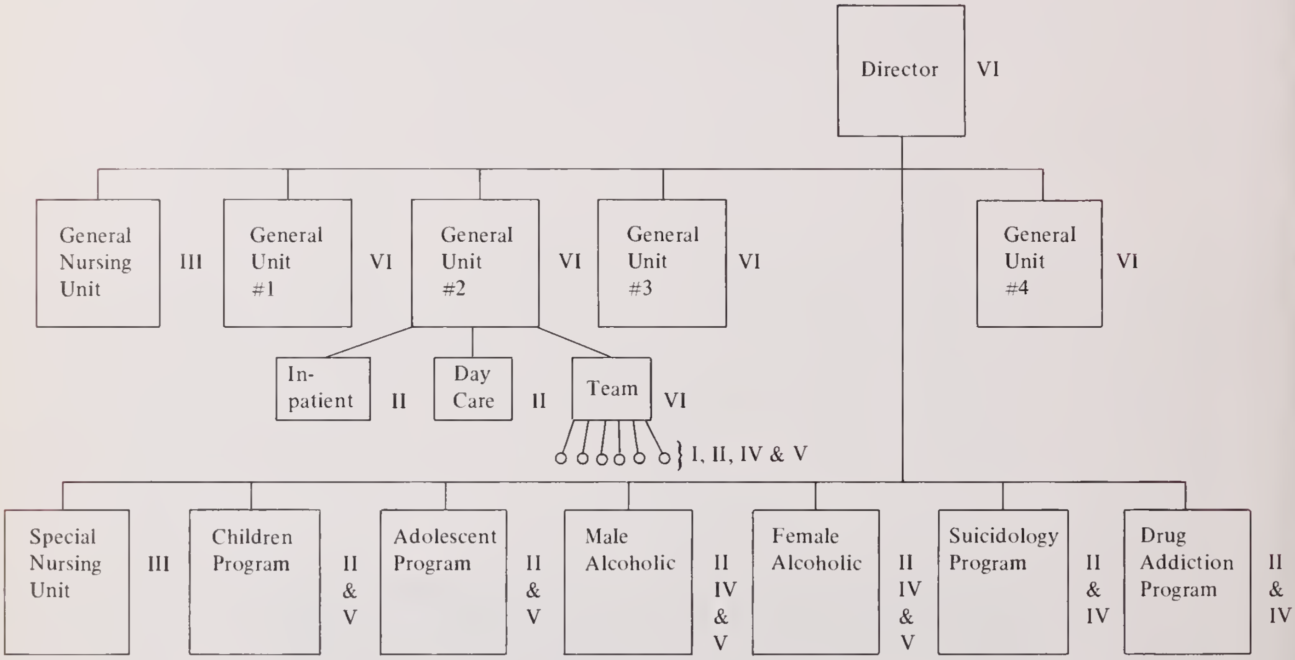
TABLE 1
Simplified Table of Organization of Area D Community Mental Health Center


Director
VI
General Nursing Unit
III
General Unit #1
VI
General Unit #2
VI
General Unit #3
VI
General Unit #4
VI
In-patient
II
Day Care
II
Team
VI
I, II, IV & V
Special Nursing Unit
III
Children Program
II & V
Adolescent Program
II & V
Male Alcoholic
II IV & V
Female Alcoholic
II IV & V
Suicidology Program
II & IV
Drug Addiction Program
II & IV

Center that shows an example of how the six models are all present in one organization. The type of model represented is shown in Roman numeral to the right of each unit.

Note that in the Area D CMHC many units have responsibilities defined in more than one way. The members of the team (the circles) usually admit by time of entry (I), but some requests are referred to a team member on the basis of a needed skill such as consultation or psychological tests (II), on the basis of specialized knowledge of a problem such as alcoholism (IV), or on the basis of special knowledge regarding a part of the population such as children (V). Note also that the unit for female alcoholics (IV, V) is also defined by modality since it is a day-care program (II).

With the six different models identified, the next question is how they should be blended in structuring mental health services. Everyone favors quality services, but we need to get past that generality to criteria that will help us determine a preference among the models. Everyone has different priorities, but objectives need to be established when structuring mental health services. One list of desirable characteristics of public mental health services is the "Ten C's":

1. Clear definition of responsibility
2. Community focused
3. Comprehensiveness of care
4. Continuity of care
5. Citizen control of major policies and directions
6. Client-oriented goal-setting for individual patients
7. Curtailment of occurrence, severity and morbidity of mental illness (prevention)
8. Comfortable and accessible entry into the services
9. Close linkages with other public institutions: schools, police, public health, judges, legislators, public welfare, etc.
10. Cost–benefit ratio the public desires and will support

Space will not allow us to detail the strengths and weaknesses of each model‡. The by-population model (especially the geographic sub-type) model has the potential of developing many of our "Ten C's". Of all the patterns, it is the one best able to meet the *raison d'etre* that we mentioned before of public mental health services, i.e., assumption of responsibilities for prevention and for care of all in need.

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‡ Upon written request, the author will supply tables on the strengths and weaknesses of each of these six models.

It might be useful to give an example to clarify why the geographic by-population pattern is superior. We are referred a 47-year-old man, last known address 1323 Main Street, who has some memory deficits, talks with looseness of associations, mentions that his wife is trying to poison him, has a history of heavy drinking, and has recently had trouble getting a job because his skills as a farm laborer are in limited demand. With the by-mode-of-delivery pattern, it can be debated whether the patient should be handled on an inpatient, day-care, outpatient, or home-visit basis. With the by-profession pattern, it is not clear whether the patient should be handled by psychologists, social workers, vocational rehabilitation specialists, or psychiatrists, since all of them, in at least some settings, work with this type of patient. With the by-psychiatric-problem model it is frequently debated whether this patient should be placed with an alcoholic program, a unit working with schizophrenics, or a sheltered work-shop program. Too often, psychiatric patients are not "pure cultures," and the by-problem model can frequently be debated. The patient's age, sex or other non-psychiatric characteristics do not point to his being segregated into any special grouping. The by-time-of-entry (admitted today) or geographic by-population (1323 Main Street) do provide a clear-cut definition of responsibility. The by-population model has many advantages over the by-time-of-entry model because it encourages linkages with other public institutions, encourages treatment in the community, facilitates a knowledge among the staff of the problems and opportunities within that client's community, avoids splitting the care of family members among several units, and encourages prevention since the person is identified as a responsibility prior to becoming ill. Empirically, this preference is supported by the two major beneficial trends that have occurred in structuring public mental health services during the past decade: geographically defined unitization of public mental hospitals, and pulling together of a variety of public mental health facilities into community mental health centers. The major advance of the 1970's may be closer linkages with other public services such as schools, public welfare, etc.; and the geographic by-population model lends itself to these linkages better than the other models.

Given a preference of the geographic by-population model, any large organization will still have a blend of all six. Too often, blending these six has been determined by the budgetary process and allocation, pressure groups, academia, professional societies, personalities, public relations defensiveness, and histories of the various components. The major deficit of these various influences is that the major purpose of public mental health service may get divorced from one's thinking in proposing an organizational structure. Some guidelines, however, can be proposed in synthesizing the six models that will allow for a more judicious determination of public mental health structuring.

As an initial step to developing knowledge on the best way to blend these six models, the following guidelines are suggested:

1. Determine the quantity and quality of the needs of those to be served.

2. Determine the quantity and quality of the resources available to meet those needs.

3. Begin the proposed organization at the top and use the by-population, geographically-defined pattern, where practical. Defining the various geographic boundaries of each unit involves weighing political, police, welfare, school and census-tract boundaries; considering the transportation system; noting the location of mental health facilities, etc. Spread (number of units reporting to Director) is a function of the degree to which the units established can function autonomously—the more autonomous, the wider the possible spread. The wider the spread, as a rule, the more services that can be provided.

4. If a needed service cannot be provided because it is too costly to divide that particular service into geographic units, then that service should be pulled out of the geographic units and a separate unit established. This usually is expressed in a high staff-to-patient ratio because the particular task requires an unusually high staff-to-patient ratio, or an expensive facility, or the number of potential clients is quite small. (Examples: 1. Because hot lines should be open 24 hours a day every day, a single unit—considering the frequency of the calls—might be preferable to each geographic unit having one. 2. Maximum security is a modality often too costly in terms of facilities and staff-to-patient ratios to expect each geographic unit to provide this service. 3. There may be a need to separate out a children's residential unit, yet the number of children to be served is too small to make it practical to staff several units.) One way to resolve debate about what services are too costly is to ask the geographic units which services they would be so eager not to provide that they would be willing to give up staff positions to have a separate unit created.

5. If a professional group has too few competent professionals to give each unit at least one competent professional of each of the needed specialties, then consider pulling out that professional group from the geographic units and establishing a separate unit. (Examples: 1. Only one neurologist as a consultant is needed. Rather than assign him to any one of the geographic units, assign him instead to the Director. 2. One finds that

he has 24 social workers to serve seven community mental health centers, but only three of the social workers are fully competent as psychiatric social workers. These 24 should form a separate unit reporting to the Director until competency is developed to where one has at least seven fully competent social workers.)

6. Following these steps should resolve which units will report to the Director. Now go into each of these units, if asked, and go through the same process in establishing sub-units, and so on. As one repeats this process throughout the organization he eventually reaches the point where it becomes impractical for units or individuals to handle adequate coverage by themselves and he then uses the by-time-of-entry model. Eventually, there is usually a point at which logistically the inpatient activities have to be defined as a unit. Eventually, there comes the point at which specialization by a unit, or at least by an individual, is reached for problems such as alcoholism and for certain patients such as children. Thus, the complete proposal usually contains six models.

7. Establish that each patient begin in a geographic unit, if practical. If not practical, select patterns for the "front lines" that still tend to have clear definitions of responsibility (age, sex, and time-of-entry patterns, for example). Flow from the geographic units to separated-out ("special") programs should be because therapists and clients see the move as therapeutic. Such a move should in no way be a "dumping." (The corollary of this is that the special programs should always be at least as attractive, if not more attractive, than the geographic units.)

These seven guidelines provide an initial skeleton on which to hang further experiences in structuring public mental health services. Using these guidelines to develop proposals will also highlight additional staffing and facility needs and answer questions as to the degree of decentralization that is desirable.

Conclusion

In analyzing the clinical structure of public mental health organizations, one finds six different models. Most, if not all, of these models will appear in any large public mental health organization. Each pattern has its strengths and weaknesses which must be considered when blending the six models. The by-population model represents the basic way to approach the major objectives of public mental health services. By using the by-population model as the basic one, guidelines can be listed to synthesize all six patterns into proposed public mental health organizations. Some of these guidelines might be considered when organizing not only public mental health services, but other large medical organizations.

Are Lower Class Children (Black and White) Inherently Inferior in Intelligence?*

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A major concern of the current research study was the theoretical formulations of Arthur Jensen,[1] in which he proposed two general types of learning or cognitive ability which are differentially exhibited by children of various socioeconomic status (SES) levels. Jensen and others have repeatedly found that lower SES groups of children score approximately 30 IQ points below the mean IQ of middle SES children. In his earlier articles Jensen assumed that these large social class differences in IQ were primarily a function of test bias rather than inherent intellectual differences between the children of the two SES levels. He thought that the tests were designed to tap the kinds of intelligent behavior acquired by children raised in middle-class environments. The conventional IQ tests were seen as inadequate for measuring intellectual potential among lower SES *Ss* and were certainly not considered to be adequate measures of the kinds of intelligent behavior developed in the lower SES environment. Jensen was, in part, responding to the recurring reports of teachers who had worked with black and white children from all SES levels. It appeared to these teachers that lower SES children were often as bright or brighter than middle-class children in non-academic areas, in spite of the fact that their measured IQ was extremely lower. For instance, they learned the names of their classmates very rapidly; they often mastered the rules of playground games more quickly than middle SES children. Conventional IQ tests appeared to miss intelligent and adaptive behavior of this nature. Jensen attempted to objectify these observations by devising tasks which were orientated to those processes of learning and intelligence which were not approached in conventional IQ tests. He employed tests which measured associative learning ability. They aimed at calibrating how fast a child could learn something new and unfamiliar right in the test-taking situation. The child's performance presumably did not depend on what he or she had already learned, which is frequently the case for convential IQ tests. The children were simply given something to learn under conditions which permitted researchers to measure the rate and thoroughness of learning. The learning tasks were designed to minimize the necessity for academic skills like reading, vocabulary, and popular facts. When Jensen employed these tasks in his research on SES and cognitive ability he found that the reliable SES differences which emerged on popular IQ tests (30 IQ points) did not emerge (no statistically significant SES differences) on his laboratory tasks. The tasks Jensen employed were the classical associative, verbal learning tasks (e.g., serial rote learning, digit span, free recall, etc.) except that pictures of objects were used as stimulus material rather than words.

Jensen initially concluded that he had discovered a culture fair method of assessing learning and intelligence. However, in his ensuing research he found that the typical SES differences which emerged on IQ tests also emerged on his "culture fair" laboratory tasks when something more than simple associative memory was required. These laboratory tasks involved conceptual and problem-solving ability. He was unable to reconcile the fact that the SES differences did not emerge on one type of "culture fair" laboratory learning task (associative learning) but did emerge on a second type of "culture fair" laboratory task (conceptual learning). Jensen[2] at that point made an exhaustive literature search in the area of intelligence (IQ), heredity, and SES. Using an elaborate set of statistical analyses, he concluded that 70-90% of the systematic variance (non-error differences) on intelligence tests was a function of genetic inheritance, while the remaining 10-30% of the systematic variance was primarily attributed to determinants present within the prenatal environment. Re-

Key of Abbreviations

SES = socio-economic status
Ss = subjects
LI = level I
LII = level II

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turning to the conflicting information regarding SES differences on his "culture fair" laboratory tasks, he proposed that he had tapped two genotypically distinct factors of cognitive ability. He labelled his associative learning tasks as measures of Level I (L I) ability. This ability was distinguished from the ability tapped by his conceptually oriented laboratory tasks which he labelled L II ability tasks. L I ability was described as randomly distributed in the general population. L II ability was proposed to be differentially distributed in the general population as a function of social class.

Jensen assumed that both his L I and L II laboratory tasks were "culture fair." He integrated his laboratory findings with his research regarding IQ, SES, and heredity, and concluded that the SES differences which emerged on the latter type of laboratory tasks (L II) were a function of inherent differences represented within the two SES subpopulations. The statistically significant SES differences which emerged on both the convention IQ tests and the "culture fair" L II laboratory tests were not interpreted to be a function of test bias but rather a function of an inherent deficiency among the lower class in conceptual ability. About 70 to 90% of the SES differences on conventional L II tasks (IQ) or L II laboratory tasks were concluded to be a product of genetic antecedents. Similarly, the common finding that black children score 15 IQ points below white children was interpreted as a function of genetic factors.

To recapitulate, Jensen made the following theoretical formulation based on his years of research: There were two genotypically distinct types of learning and cognitive ability. L I ability was simple association and rote memory and was unrelated to SES. L II ability, whether measured by "culture-fair" laboratory tests or conventional IQ tests, involved abstract or conceptual thinking. Lower-class children as a group were born with a substantially lower capacity for L II or conceptual functions. This was the reason for the large social-class differences emerging on L II measures, regardless of their culture fairness.

Jensen has, on several occasions, cited the results of Glasman's doctoral dissertation[3] as a major piece of supporting evidence for his two factor theory of intelligence. Glasman found that groups of lower and middle SES children differed just as significantly on a "culture-fair" L II free recall task (recall pictures that could be conceptually categorized) as they had on a conventional IQ test. His own research indicated that they did not differ on an L I version of the task (nonconceptual). Jensen interpreted spontaneous conceptual classification on this task as a prime manifestation of L II or abstract ability and pointed out that middle SES *Ss* enhanced their free recall on this basis, while lower class *Ss* did not.

Jensen's L II laboratory tasks represented some of the most convincing evidence for the genetic explanation of SES differences because they were assumed to be "culture fair" and unconfounded by previous learning. Lee J. Cronbach[4] took issue with this interpretation. He proposed that SES differences on L II laboratory tests like Glasman's were very probably a function of something less than genetic differences. Cronbach predicted that low SES *Ss* would show substantial improvement on L II laboratory tasks with a minimum of pre-training in conceptual classification. Furthermore, he predicted that SES differences in L II ability would wash out with pre-training. In a reply to Cronbach, Jensen[2] implied only *Ss* with high L II capacities would profit significantly from pre-training of that nature. Only *Ss* with high L II capacities would be able to transfer pre-trained conceptual skills learned in one situation to a new and unfamiliar situation which required *Ss* to use the pre-trained skills in a different way (e.g., on an L II free recall task). Lower class *Ss* would not be expected to make a substantial transfer because they were L II deficient. Secondly, Jensen proposed that SES differences in L II performance would become accentuated if both middle and lower SES *Ss* received equal portions of pre-training. The gains made from pre-training would be a geometrically expanding function of initial, L II ability. Both groups might make some improvement, but the middle SES group would make the greatest gains as measured by a post test.

The diametrically opposed predictions of Cronbach and Jensen were tested in the current study. A different approach to assessing conceptual ability was introduced. Glasman's L II free recall task involved conceptual classification. Other researchers[5-6] had investigated this form of cognitive activity from the theoretical viewpoints of Piaget[7] and Bruner.[8] They employed non-verbal tasks which measured a child's capacity for multiple classification and multiple seriation from a developmental perspective. These tasks were currently used to identify lower SES *Ss* who did poorly on Jensen's L II tasks but nevertheless exhibited a high level of conceptual ability. It had been assumed, on the basis of Cronbach's arguments, that many lower SES children had well-developed conceptual abilities and could be readily made to manifest these skills on more verbally oriented conceptual tasks (e.g., Glasman's) after pre-training.

The aforementioned issues led to the formulation of three empirical questions:

1. Does pre-training in simple concept formation positively influence the performance of typical low SES children on more complex conceptual tasks (e.g., Glasman's L II free recall task)?

2. Does pre-training in simple concept formation influence SES differences in L II performance?

3. Does the performance of typical, low socioeconomic children on multiple classification and multiple seriation matrices predict the outcomes of the first two empirical questions?

The procedure of the current study involved three phases. During Phase I, 368 fourth graders were passed through a series of selection sieves. A pool of 87 middle SES *Ss* and 123 lower SES *Ss* was finally selected. They represented the modes of the two SES subpopulations which typically emerged in Jensen and Glasman's research. They differed substantially in L II ability (30 IQ points) but did not differ significantly in L I ability. If Jensen's theory were correct, these groups would have represented two, non-overlapping, genotypically distinct, subsamples of L II ability.

In Phase II these samples were administered the multiple classification and seriation tasks. Approximately 96% of the middle SES *Ss* achieved maximum performance on these tasks and were labelled category A *Ss*. More than 48% of the lower SES *Ss* achieved an equivalent level of performance and were labelled category B *Ss*. while the remaining lower SES *Ss* were labelled category C *Ss*.

During Phase III, 36 *Ss* from each category (A, B, and C) were selected, for a total of 108 *Ss*. Category B and C *Ss* were equivalent on the initial L II ability measure and 28 IQ points below the category A *Ss* on that measure. None of the samples differed on the initial L I measure. The *Ss* from each category were then subdivided into two subgroups of 18 *Ss* each which were equated on L II ability, sex, race, and age in months. One subgroup was randomly assigned to the experimental condition. The experimental *Ss* were given two, 20-minute sessions of pre-training in concept formation. A few days later they were administered Glasman's L II free recall task without any indication to experimental *Ss* that their pre-training was a preparation for this task. The items used in training were different from those comprising the free recall task.

Dr. Scrofani is shown above receiving the first annual Overholser Prize for his paper on the intelligence in lower class black and white children at the spring meeting of the Medical Society of Saint Elizabeths Hospital. The prize of $50.00 is being presented by Mr. Clark S. Aist, Acting Associate Director for Non-Medical Training, Overholser Training and Research Division. The Overholser Prize attempts to recognize and encourage outstanding and creative contributions to the broad field of mental health by trainees of Saint Elizabeths Hospital in the non-medical behavioral sciences.

A 3X2 analysis of variance design was used to statistically analyze the data. There were eight indices of L II performance based on the responses of the L II free recall tasks. Statistically significant interactions emerged for six of the eight L II indices. The nature of the interactions was statistically investigated with a variety of post tests.[9] Pre-training had a positive and statistically significant effect on the L II performance of category A and B *Ss* but not for category C *Ss*. It was also found that in the control condition category A *Ss* were significantly higher than category B and C *Ss*, who did not differ significantly. However, in the experimental condition category A and B *Ss* did not differ significantly but were both significantly higher than category C *Ss*. Therefore, pre-training had a differential effect on category B *Ss* in a positive direction, as compared to category A *Ss*. Specifically, category B *Ss* were significantly below category A *Ss* in the control condition but not in the experimental condition, indicating that the gains made by category B *Ss* were greater than those of category A *Ss*. Finally, post hoc statistical analyses were performed on the variable of race, as a matter of secondary interest.[10] No consistent patterns involving race differences emerged in the current data, an additional finding that was not in accord with those of Jenson.

The following conclusions have been drawn from the findings of the current study:

1. Pre-training in concept formation has a positive and significant influence on the L II free recall of a substantial portion of lower-class children who exhibit IQ's that are modally typical of their SES level.

2. Socioeconomic differences on L II free recall are significantly reduced as a function of pre-training.

3. Performance in multiple classification and multiple seriation predicts the outcomes of the first two conclusions with respect to typical low SES children.

At a broader level, the current findings supported the assumptions and predictions of Cronbach, while the theoretical assumptions of Jensen were counterindicated. Many lower SES *Ss* (category B *Ss*) effectively transferred conceptual operations learned in one situation, to a new and unfamiliar problem situation. Social class differences between category A and B *Ss* were significantly diminished rather than accentuated by pre-training in conceptual thinking. More importantly, those in category B *Ss* represented over 48% of the current lower SES population of children who would otherwise be identified as L II deficient by Jensen's laboratory techniques. A substantial portion of the laboratory evidence which has been cited to support Jensen's genetic hypothesis was thrown into question by the current results. I am presently involved in two additional studies which are aimed at critically questioning other portions of Jensen's theory under controlled conditions. The support for Jensen's genetic hypothesis which he has gleaned from identical twin studies remains untouched by the current study, but contradictory data and alternative interpretations regarding that area of the research domain has been appearing in professional journals with increasing frequency.

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Thinking About Schizophrenia*

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The central mystery of psychiatry is the phenomenon of schizophrenia. We do not know whether we should consider schizophrenia a disease, or if a disease, whether one or many. When speaking of schizophrenia, let us also not pretend that we are talking assuredly about a condition with a clear definition, but rather about a mystery the general contours of which we more or less understand. Let us also admit that when we speak of a schizophrenic person we are speaking of a human being who has acquired a name, but we are not too clear about the naming process. We are talking about someone we are in the habit of so naming, probably for excellent reasons, but yet reasons not fully and consciously known to us.

As years pass and one studies and treats these schizophrenic people, the sense of mystery deepens. No theory or hypothesis does more than touch the edges of the mystery. Our patients are strange and tragic people, and although a profound rapport can develop between us and them, we remain strangers in the end, for the rapport be sustained without an odd and painful sense of strain. It is quite as if we and they existed on different planes of consciousness, which can intersect only fitfully and with regard to certain limited areas of experience.

We possess almost overwhelming evidence of an inheritable but entirely unidentified biological issue in schizophrenia, and fairly convincing evidence that among the environmental issues those involving role confusion may be most critical.

Many of you are now saying privately, "that fellow is living in the middle ages. Schizophrenia, whatever its etiology, is now an outpatient disease, the patient is in vastly better shape than before, his thinking disorder minimal, he lives, breathes and talks more or less like a human being, and if you treat your patients as if they were so different from yourself, that in itself is the very source of the difference. Expect them to be schizophrenic and they will be. They are indeed sick, passive, suggestible people but not in any special sense different."

I know that these and many other related thoughts are going through your minds. To parry them I shall make a few rather brutal remarks. If we examine the actual quality of the lives of these people we now return so easily to the community, in their homes and foster homes and halfway houses, taking their Thorazine tablets or their Prolixin injections, we will discover that the great majority, while outwardly sane and tractable, are living utterly barren and blasted lives. If they work, which is not likely, their work is drab and menial, and probably depends on a paternalistic employer. If they play, which is not too likely, it is as if they were automata. If they read, it is the comic page, if they speak to each other it is about cigarettes or food. If they make love, which is very unlikely, it is mechanical and very infrequent. They give little warmth, and what they get from others is at best good-natured patronizing tolerance. They are not happy, nor are they sad. In truth, they are barely alive and exist as men and women in commdemned cells. We have created a kind of slow spiritual euthanasia with chemical agents, whose primary function is to get the patients away from us so that by not seeing the poverty of their lives, we may cease feeling any responsibility in the matter. Indeed, we are simply concealing from ourselves the nature of the problem. What we have achieved in relation to this great and wasteful catastrophe of schizophrenia is at best mild amelioration, and there has been a price exacted.

Our responsibility as physicians is to continue thinking about a mystery that we fail to grasp and transform our thoughts into actions that may deepen our understanding or increase our healing powers. Thinking clearly about any problem as strange and perplexing as this requires a kind of preparation. The first step is to try to suppress, as best we can, preconceptions that block and impede inwardly free inquiry. One such preconception, although a fairly mild one, is an excessive certainty that the conflict model of emotional illness is always and necessarily true. Another and more serious one is that we are not free to think or talk about the problem of consciousness, because of

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the hold of mechanistic thinking dominant in science and medicine since the seventeenth century.

The belief that consciousness is not a proper subject for scientific inquiry is sheer ideology, not an ingredient of tested scientific knowledge. We must think about consciousness because, far more clearly than in any other disorder, the most impressive aspects of the whole problem of schizophrenia are inextricably bound up with the problem of consciousness. I believe schizophrenia to be a disorder of consciousness, or more precisely, a disorder of the processes or entities responsible for consciousness. At this juncture, the part of the audience I have not already estranged may very well part company with me altogether. I can only beg of you to suspend judgment a while longer, and at least acknowledge that I have given fair warning as to the direction in which I am heading.

The next preparatory task is that of simplification. We cannot think clearly about a problem in a diffuse way. We must try to get at the heart of our mystery, or at least at a definition of our mystery. Eugen Bleuler was the first great simplifier of the problem of schizophrenia, and we must turn back to him for a clear initial statement of our problem.

Bleuler spoke of the splitting of mental functions as the central descriptive feature of dementia praecox. In contemporary language, we would say, parapharasing Bleuler, that schizophrenia is a condition involving a pervasive failure of integration of behavior and experience, rendering the person unable to cope with the demands inherent in the social roles existing outside of institutions. This failure of integration takes many observably different forms, and Bleuler did not insist on any specific manifestations. His conception of schizophrenia was a good deal more subtle than our medical school version.

Can we sharpen Bleuler's description? Let us consider the phenomenon of loosening of associations, which Bleuler thought to be the most important single manifestation of integrative failure: The dislocation of trains of thought in schizophrenia can be best described as a failure to maintain a fixed goal over a time interval long enough to permit adequate communication. A sentence begins on one topic and ends on another. There may be a clear discernible thread or purpose to the conversation, but the means employed are seriously fragmented. This is a failure to integrate in the dimension of time. Recent memories of one's own thinking and behavior, present actions, and anticipated future are not welded together in the relatively coherent unity found in most conversations. The schizophrenic is anchored in a smaller moment of time, his past is too short and his future almost nonexistent. He drifts on an eternally present moment. This must be the simplest way to explain one of the most perplexing incidental features of schizophrenic living, the absence of boredom. Boredom requires a sensation of time passing, a lack of contrast of past with present. If the sense of passing time were to disappear or contract to a small moment, boredom would become impossible. This seems to be the case with the schizophrenic. We are not imputing to the schizophrenic a failure of memory, rather a failure to integrate recent memories that we do know to be available to him, at least as fragments. This failure to integrate more than a small stretch of time also accounts rather easily for the remarkable dearth of more than short-term goals in the patient's behavior.

In like fashion, we can look upon ambivalence as the same kind of failure to integrate emotions and impulses across time. Hostile and friendly gestures may follow each other in the same disconnected fashion as the contents of speech. The idea of loosening can be applied to non-verbal as well as verbal behavior. Ambivalence seems adequately accounted for as another instance of failure to integrate a sufficiently long span of time. A train of impulses or feelings is not synthesized or fused into a compromise, as in the case of normal or neurotic ambivalence. In the latter instance, the presence of opposing trends must be inferred rather than directly observed, as it may be in schizophrenic behavior.

The failure of abstract attitude in the schizophrenic is somewhat harder to account for, but a case can be made that a generalization from a set of similar objects or events is a sequential process in our thinking. We really do not simultaneously grasp a set of related things but go from one to one in a series to develop a covering idea. Schizophrenics have plenty of ideas but they are badly related to their experience. I believe that explaining the inability to generalize well as a failure to organize a series of cognitive acts in time makes more sense than using failure of abstract attitude to explain the other deficits. In any case, the idea of a failure of temporal integration covers the descriptive ground better than any other single idea. We are supposing that the schizophrenic is not living in as long a present moment as the rest of us. Virtually every aspect of his adaptation can be related to this apparent fact. We are not obliged to assert as yet whether or not he is unable to expand his present moment.

It must be pointed out that this failure of integration across time is not a fixed thing in schizophrenia. It is worse when the material to be integrated involves emotions of any appreciable intensity, or complex issues of personal identity. Warm personal contact over a period of time, moderate use of tranquilizing agents, and well-structured environmental situations all act to strengthen the slender resources of the schizophrenic, but he fares badly if he loses these supports even temporarily. With this qualification in mind, let us tentatively adopt a single descriptive principle. It is at least as good as any other, and satisfies our demand for simplicity in thinking about our problem. The person we call schizophrenic or one predisposed to schizophrenia has a severely limited capacity for organizing his experiences in time. William James coined a phrase, "the specious present," to express the fact that in normal consciousness the duration of the present is not the infinitely short moment of physics but a finite stretch of time. We are aware of more than an instantaneous moment, since otherwise we would not be able to perceive movement. The implications of the past and future are with us, and the temporal context of our activity is of many minutes, hours, or even years. We are supposing that the specious present of the schizophrenic is much shorter than that necessary for normal adaptation, and this results in a collapse of much temporally integrated behavior.

If we are to be somewhat more precise, we must add that for very short-time intervals, such as the time it takes to light a cigarette, hit a pitched ball, tie a shoe, or make a quick witticism, the schizophrenic is absolutely unimpaired, immediately distinguishing him from the usual varieties of organic deficit. Very short-term integration is fully intact. This is very strange, since one would suppose the same neural systems to be involved.

Our hypothesis, while it presupposes a drastic alteration in the experience of time, contains only a quantitative distinction between normal and schizophrenic functioning, namely, the span of time over which integration is occurring. We communicate with the schizophrenic only when we share with him experiences which are essentially momentary, a feeling with no beginning or end, or a short-range purpose. He cannot share with us our increased duration of specious present, at least not fully, and we cannot share with him an autistic fragment which fills its context completely.

Is this the inheritable organic defect of schizophrenia or is it a complex consequence? It is apparently a labile deficit, as indicated by the occasional capacity for marked improvement at any stage of the illness. Is it possible that the patient has chosen or learned to constrict his specious present, to avoid some special unpleasantness in the experience of time? Let us avoid the easy answer that he does so to avoid anxiety. If that were so, it would be an occasional affair, occurring when anxiety loomed as a nearby experience, as in neurotic symptom formation. It would not occupy half a lifetime in an unrelenting way. Could there be a non-psychological unpleasantness or a kind of unpleasantness not labelled in our psychological vocabulary? We cannot escape the deep conviction that willfulness is inherent in schizophrenic living. It is impossible to be satisfied with an explanation that represents the schizophrenic as only a passive victim. Those of us who work closely with these people know that this is not true. I am hinting at a willful shrinking of the specious present. It seems a little fantastic but it does go a long way as an explanatory idea. Let us play with this further. Why might such inner behavior occur? Is there something disagreeable as we have suggested in the sensation of passing time, something that if intensified would lead to such a contraction of consciousness as an adaptive measure?

Well, of course, the schizophrenic has been through a ghastly experience, the experience of more or less acute psychosis, when the contents of his mind literally ran riot. He was overwhelmed by experiencing his associations running out of control and exploding into a waking nightmare. Once again we run into the same basic problem of integrative failure, but we gain some information. The associative trains to recent experience cannot any longer be smoothly integrated, but the riot of associations was ended by shrinking the specious present. These associative trains are the reverberation of just-past experience, now recent memory. The schizophrenic, at a certain point in time, could not maintain the integrity of this system and retreated into a span of time that he could handle. This, of course, does imply a rather fantastic power of suppression or disengagement.

Notice that as we develop this idea we are forced to conceive of certain mechanisms. One is a mechanism of inclusion, that is, a mechanism which establishes the boundaries of the mind and the extent of awareness, but which is distinct from those mechanisms responsible for the unification or integration of the enclosed materials. In the early evolution of schizophrenia we see an increased volume of inclusiveness, a period of acute disturbance, with vivid rapid thought processes and intense flashes of rapport with others. In the transition to stable chronicity we see a shrinking of the size or scale of the self, which we are now measuring in units of time, to a scale that can be successfully integrated.

We should pause for a time to sketch out the requirements for a theory of integration. Integration of mental or behavioral events means both the imposition of a form upon a collection of events and the transformation of unruly elements among those events so that they fit in or dovetail with the others. The purest and indeed most remarkable instance of such integration is the construction of a finished dream. We must accept Freud's concept of secondary elaboration as simply factual. The dream is organized in such a way as to form a coherent whole, acceptable to consciousness, its absurdities smoothed over as competently as in a finely constructed play. The final touches of coherence extend into details of perspective and coloration that are astonishing, for a whole scene never before beheld is rendered exquisitively well by an organizing process of almost unbelieveable power. It is as if some intelligence had gotten hold of the actions of myriads of neurons and coerced them into the generation of coherent form which dovetails in all its elements. This same coercive power of intelligence seems operative in the handling of recent memories and their evoked trains of association. Although a less dramatic performance, the maintenance of steady goals and themes in thought and behavior requires the same kind of inner coercive power acting upon the associative trains evoked by our recent experience. The immense organizational strength of mind represents a mystery deeper than schizophrenia, since an understanding of its nature would lead to an understanding of its aberrations.

One question leads to another, and it almost seems as if the solution to the problem of schizophrenia requires a solution of the problem of consciousness, the most conspicuous failure in man's whole long intellectual history. Let us hope that some more modest and limited questions can still be useful, although we must not be afraid of strange speculation, for if anything is certain it is that the eventual explanation of schizophrenia would seem to us now to be utterly mad if it were presented to us even with supporting data. Let us face the likelihood that all sensible and respectable theories of schizophrenia have been advanced already and that none of them work.

We might entertain the idea of a kind of gate which excludes recent memories older than a certain duration, so that the integrative actions of mind are restricted to the very recent past, at least most of the time. Since memories do seem to be dated relative to the present, an apparatus might be conceived which blocked entry into awareness of all events more than a few seconds old. The trouble with such a conception is that such a gate would have to be of very elaborate construction, since surely these memories have no clear anatomical segregation. We require of mind the same kind of searching and detailed suppressive power that dreaming would also required if we think of the brain as a conventional machine. Perhaps our whole point of view about the relation of physiology to mind is wrong.

At this point I come to a complete halt if I try to pursue this inquiry any further in a logical and methodical way. When logic fails we must allow imagination to take over and not be afraid of unconventional ideas. I shall try one out on you and hope that you follow the example and consider alternatives without the fear of embarrassment.

Let us suppose that Williams James' specious present is literally true, that is, let us suppose that the subjective present has a true duration in time without a sharp beginning or end. As conscious entities we exist in this duration of time, where a definite interval is for us simultaneous, not a succession. We are in the habit of supposing that our integration of recent past with the present is the integration of recent memory-traces with immediately present sense-experience. I am proposing a slightly wilder idea, that we are genuinely still in the recent past, or rather what is definitely past to the physicist is to us really and literally present, and possibly a bit of the future too. We are *in* time. I believe there are some strong philosophical arguments for such a view, which have nothing to do with psychiatry. Remember, that because of Albert Einstein we are permitted to think about time in this general fashion without contradicting contemporary natural science. Our only concern here, however, should be whether such an idea is useful in thinking about the schizophrenic alteration of consciousness. It does immediately provide us with a simplier formulation. Suppose that indeed there is a mechanism, not of a kind we can readily visualize because we do not have a four-dimensional imagination, but nonetheless an actual mechanism which determines the length of our literal specious present, a viewing slit as it were whose width is determined by some unknown dynamic process, in even more concrete language, a mechanism which establishes the thickness of consciousness in the direction of time. Suppose further that this mechanism's stability is related to the amount of mental traffic it bears, the amount of mental information it contains, if you will, remembering that mental information is a dynamic flux at all times. If this process were to become unstable, so that the duration of time it contains were to become an oscillating variable, presenting to awareness a larger mass of information than any human nervous system could integrate, and then contract until some final equilibrium were reached, we ought to see something very like the evolution of schizophrenia. We would indeed have a very compact hypothesis about the disorder, which would be consistent with the lack of integrative deficit in short-term goal-directed behavior. Our emphasis has shifted away from integration to the mechanism of inclusion or boundary setting that we spoke of earlier.

We would be obliged, however, to begin thinking about biological systems in a four-dimensional frame-of-reference, but we would also gain the advantage of a much more natural formulation about consciousness in general.

As a final question we might consider the possibility that this four-dimensional process is inherently social rather than individual and that it has an unconscious collaborative aspect. This would help to explain the collapse of the subjective present to such a small compass. The schizophrenic person, who has experienced the catastrophic instability I have described, arrives at an adaptive solution which rejects the social process and must therefore reject the deep supports of that process. We might even speculate that the normal duration of consciousness is a collective product in a jungian sense. But at such a point we have to stop because we simply do not have the conceptual equipment to continue.

Whether or not we stay with the conventional view, the idea of a contracted specious present is not a useless abstraction, as it has already led me to re-formulate both research and treatment plans. The role of speculation in science is its power to suggest experiments, as Claude Bernard understood so clearly. One set of possible experiments is very simple. To present stimuli in a sequence which would generate a whole or 'gestalt' if they were perceived simultaneously, slowly spelled words, or slow-tracing of shapes by a moving point of light, are examples of such presentations. By such techniques we might establish whether or not there is a genuine failure to utilize recent memory traces. The same procedure could be expanded into a treatment device.

The deeper question of the possible reality of the specious present has also suggested some experiments concerned with the statistical analysis of short-term behavioral data, but the details are far from clear.

In closing, I hope you will not seriously misunderstand me. I am not trying to convince you of any particular exotic point of view. I am, however, trying to insist that when we are totally at a loss to understand something with our current intellectual conventions, we must search for radically new points of view and let our imaginations roam. We must not be afraid to appear eccentric or cranky, since all progress arises from disciplining of fantasy.

Mourning Precipitates Porphyria

DAVID L. FEDDERS, M.D.*

Among the multiple factors known thus far to be involved in the dynamics of an attack of acute intermittent porphyria (AIP), there is no direct documentation of the role of psychological factors as etiological. A case is presented in which the specific psychological process of mourning preceded attacks of porphyria.

Report of Case

A 33-year-old colored woman was admitted to a general medical hospital for the second time for an attack of AIP, hepatic type, characterized by symptoms of colicky abdominal pain, nausea, vomiting, and constipation.

She is an only child. She describes her father as irresponsible and alcoholic, and her mother as quite submissive until alcohol became such a problem with her parents' marriage that it ended in divorce. The patient's husband was also alcoholic, and her marriage ended at the age of 26 with her retaining custody of their four children, the youngest of whom was a neonate. It was after the birth of her youngest that the diagnosis of AIP was established, partly as a result of the peripartum complication of hypertension.

She has since had yearly episodes of abdominal pain occurring usually in October, November, or December, not requiring hospitalization. These were treated conservatively with bed rest and analgesics.

At the age of 28 she was hospitalized for the first time with symptoms of abdominal pain, nausea, constipation, malaise, muscle weakness, and aphonia. This occurred two weeks after the death of her 22-year-old niece from pulmonary carcinoma. Her niece was her mother's only sister's only daughter, and was quite close to the patient. "She was like a sister to me, since I was an only child." The patient had encouraged her niece to see a doctor about a "boil on her buttock" in order "to get it lanced." The lesion was metastatic carcinoma, and the niece died within three months.

The patient recovered from the porphyric symptoms and was relatively asymptomatic except for abdominal pain at the year's end, until the second and present hospitalization, which was preceded by the death of her maternal grandfather one week prior to admission. She spoke fondly of visiting grandfather as a child, spending summers there and traveling by train "all by myself" to see him. He regarded her as his favorite and she thought him "a better man than either my father or husband." He died from a hypertensive cerebral vascular accident.

After his death the patient felt depressed, "I felt like dying myself because all my friends were gone." She was tearful, sad, and anorexic at times during her hospitalization but was thought to be in mourning rather than clinically depressed.

Discussion

Since the first classification of the porphyrias by Günter in 1912[1] and the first description of AIP as a clinical entity by Waldenström in 1937,[2] much work has been done in the way of etiology. The most basic defect of the disease, acute intermittent hepatic porphyria, is that it is a mendelian dominant genetic defect characterized by functional hyperactivity of the enzyme δ-aminolevulinic acid (ALA) synthetase and consequent overproduction of porphobilinogen (PGB) which when released into the urine and oxidized by light, causes "alicante-colored" red urine. The symptoms of the clinical disease seem not necessarily to correspond with PBG overproduction. The symptoms are broadly classifiable into two groups: 1) gastrointestinal, including abdominal pain, nausea, vomiting, constipation, and diarrhea and 2) neuropsychiatric, including nearly all functional neurotic and psychotic symptoms and nearly all symptoms of paresis of peripheral autonomic and voluntary nerves of the central nervous system.

Factors known to precipitate attacks of AIP fall into four general categories including the following:

1. Drugs–barbiturates, sulfonylureas, gresiofulvin, alcohol,[3] hexachlorabenzene,[4] and chloraquin[5]
2. Metals–iron overload[6]
3. Exogenous hormones–estrogens, steroids, and anti-fertility agents[3]
4. Physiological changes–fasting[7] and variations in menstrual cycle[3]

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In the case presented there was no history of taking any medications, vitamins, or anti-fertility agents, and eating habits were regular. The two most significant attacks in the life of the patient occurred in association with the loss of a significant love object and the corresponding mourning process. No doubt there were experiences of loss which did not lead to attacks, such that this experiment of nature lacks an adequate control. However, the temporal relationship of mourning to acute attacks in this person indicates a need for further examination and observation of the psychological aspects of porphyrias, in order to evaluate the strength of psychosomatic factors in a multiple etiologic approach to porphyria.

In view of this approach one would wonder to what extent depression causes insomnia which leads to the use of barbiturates as hypnotics. Likewise, the anorexia of depression could cause prolonged fasting. Variations in menstrual cycle, abuse of alcohol, and lowering of resistance to infection with treatment by antibiotics are all reasonable accompaniments to depression as well. One would thus wonder further about a central role of depressed affect in the development of a porphyric attack.

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Porphyria in Royalty*

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During the past several years Ida MacAlpine and Richard Hunter have succeeded in clarifying the baffling "madness" of King George III. In a series of studies culminating in a book entitled *King George III and the Mad Business*, (Pantheon, 1970), the conclusion is that, although King George III was not a well man, he was anything but psychotic. The historical appellation of "mad King George" reveals how skillfully political coloring can distort history until sufficient time elapses—in this case a century and a half—to permit facts to emerge.

The critical review of the signs and symptoms of King George III's putative "madness" combined with the unique opportunity to conduct a genealogical survey of the related royal houses, and finally the opportunity to examine the living descendants of King George III, have established beyond doubt that this much maligned King was the victim of a painful and delirious malady now known to be porphyria. His attacks were characterized by colic, constipation, nausea, painful weakness of the legs, rapid pulse, sweating, attacks of hoarseness, visual disturbances, difficulty in swallowing, intractable insomnia, excitement, dizziness, rambling, and convulsions. The physicians attending him described his mental state as follows: "Delirious all day—impressed by false images—continually addressed people dead or alive as if they were present—engrossed in visionary scenery—his conversation like the details of a dream in its extravagant confusion."

During the time of his illness the numerous attending physicians had few means of assessing his physical condition other than feeling his pulse, looking at his tongue, and inquiring about his "animal" or excretory functions. There was no stethoscope nor sphygmomanometer available during those times, nor was there even a reliable clinical thermometer. The physicians were further handicapped by royal protocol which prohibited the questioning of the King about his symptoms until he first addressed them. Thus we find in a report the following statement "His Majesty appears to be very quiet this morning, but not having been addressed we know nothing more about His Majesty's condition of mind or body than what is obvious in his external appearance." Perhaps there might be some kinship of feeling between these physicians and the psychoanalysts of today who have experienced the frustration of a silent hour.

Although the assessment of the King's physical symptoms were necessarily vague, such was not the case in assessing his mental symptoms. His mental state was the cause of such concern that numerous physicians who specialized in "intellectual maladies" were called in and took residence in the palace. The most famous of these practitioners was the Rev. Dr. Francis Willis who was called "doctor duplicate" because he was both a doctor of medicine and theology. Dr. Willis arrived with the tools of his establishment which included attendants and a straight jacket and did not hesitate to apply the same form of treatment to His Majesty as he did to any other insane person. The King's stormy and unpredictable behavior was considered to be a furious mania and his fierce dislike of doctors was considered as evidence of his delusions.

The presumed insanity of George III aroused wide public and professional interest in mental illness and brought about the first systematic attempt to deal with it as a medical problem. This led to the establishment of psychiatry as a serious branch of medicine. William Black, a teacher of medicine, aware of the fumbling efforts of the physicians to forecast the prospects for the King's recovery, began a systematic study of the question and became the founder of psychiatric statistics. In studying the records of people pronounced insane, he came to the conclusion (sometimes called "Black's law") that approximately a third of such patients recover completely, another third recover considerably, and a third do not improve or actually worsen. There has been little improvement on these statistics. Another physician, Richard Powell, observed that following the King's 1788 attack there was a considerable increase in the number of insane persons admitted to private asylums. His findings were presented graphically in a histogram which introduced this device into medical reporting for the first time. Two of King George II's sons, the Dukes of Kent and Sussex, established the first fund for research in psychiatry which resulted in the first controlled trial of a treatment for insanity. In the course of these studies a London physician named Edward Sutleffe offered a remedy which he called a "herbaceous tranquilizer," thus introducing the term that describes the dominant treatment of mental illness with drugs today. Parliament became increasingly concerned about better care for the mentally ill, especially among the poor, and set up a committee "to inquire into madhouses." The committee after two years of study provided for the system of caring for all mental patients in asylums.

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Finally, the genealogic studies of the illness show a widespread incident of porphyria throughout the Hanoverian royal house and also carried over to the Brandenburg-Prussian house via the sister of King George I. The first identification of porphyria was in Mary Queen of Scots.

In summary, we may note the historical interest of this illness occurring in King George who was so intricately involved in the American struggle for independence and whose porphyric symptoms which lasted a maximum of perhaps 3 to 6 months in his entire life of 81 years were politically recast to suggest a psychosis. In addition, the establishment of psychiatry and the funding of research in psychiatry and the establishment of a more humane care for the poor mentally ill directly resulted from the great concern for the illness of King George III. Thus, we find porphyria a "non-psychiatric" metabolic illness as a most significant factor in the development of English psychiatry as a separate branch of medicine.

Group Psychotherapy with Manic-Depressives

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This paper deals with group psychotherapy with manic-depressive patients over a period of one year in an inpatient setting. It describes the method of group psychotherapy used and some of my observations coming from my work as a clinical associate on this ward.

Setting

The setting was an inpatient research ward at National Institute of Mental Health. The patients were referred from all over the country because they were diagnosed as having either bipolar manic-depressive illness or unipolar depressive illness. All had a history of multiple hospitalizations and outpatient psychotherapy. The youngest was 28, the oldest 62. There were no more than eight patients at any time on the ward and no less than six. All patients attended group psychotherapy. The average length of stay was six months, although three patients stayed for one year. There were a total of four males and fifteen females. Approximately half of the patients at any one time were in individual therapy with the group therapist, while the other half were in individual therapy with three other therapists. The ward was close-knit with a milieu type of treatment philosophy. Among other things, such as staff and research meetings, individual, group, and family psychotherapy, there was a therapeutic community-type meeting once a week for one hour at which time out-of-hospital passes and problems were discussed.

The patients all received double-blind medication and placebo which the individual, group, and family therapist were kept blind to, as well as the nursing staff. Patients were also expected to participate in research studies varying from spinal taps to sleep EEG studies. The group psychotherapy sessions were twice a week for one hour per session. At times one session a week was video-taped. Since the ward nurses requested group therapy training, they were provided with such opportunity. Over the year there were two nurses, one for the first six months, and the other for the last six months.

Method

Before the group began, the therapist and nurse discussed how the group would be conducted with the nurse as co-therapist. Initially, because of the nurse's lack of training in group psychotherapy, she preferred to take on a secondary role and functioned as an observer. The plan was to start the group in a Bion fashion and slowly develop the group toward an Ezrielian model. Bion, a psychoanalyst, conducted groups at the Tavistock clinic in England in the late 1940's and made astute observations concerning them. He described two groups occurring at the same time, the work group and the basic assumption group. This is an outcome of the manifest and latent, i.e., the conscious and unconscious postulates expressed in psychoanalytical theory. Bion called that part of the group which was conscious, rational, reality-oriented and task-oriented, or having characteristics of the ego as described by Freud, the work group. The activities of the work group are obstructed, diverted and, on occasion, assisted by certain other mental activities which have in common the quality of powerful emotional drives. This is the emotional, instinctive aspect of the group, with little relationship to reality. This is the basic assumption group. The group members come together and stay together because of strong basic needs or assumptions. The first assumption is that the group exists in order to be sustained by a leader on whom it depends for "nourishment," material and spiritual, and protection. Bion called this mental state "Basic Assumption Dependency" and its leader the "Dependent Leader." The second basic assumption, "Basic Assumption Pairing," is that the group has met for pairing. It is suffused with messianic hopes, and its leader can best be described as the unborn genius. The third basic assumption is that the group has met to fight something or run away from it. It is prepared to do either indifferently. Bion calls this "Basic Assumption Fight-Flight" and its leader the "Fight-Flight Leader." Bion's interpretations are solely group-centered and made only to the group as a whole.[1-5]

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Ezriel was a psychoanalyst working with groups who further refined Bion's theories. In place of Bion's basic assumptions, he postulated an underlying common group problem that gives rise to tension and is a common denominator of the dominant unconscious fantasies of all group members. Ezriel speaks of the common groups' tension, the covert affect which is shared by all members of the group. He describes three kinds of object relations. The first is called the required relationship, in that the patient tries to establish it with the therapist to avoid the second, the avoided relationship, which he feels he has to avoid in external reality because he is convinced that if he gives in to his secret desire of entering into it, this would inevitably lead to the third relationship, a calamity. Ezriel emphasizes interpretation of the here and now experience, which is simultaneously group-centered and individual-centered. His interpretations are group-centered in that they first point out the unconscious common group tension and resultant structuring of the group and are individually centered in that they pinpoint the common contributions of the individual group members to this group tension. Ezriel believes that the maximally effective interpretation identifies the three aspects of the current emotional situation in their relationship with one another, the required relationship, the avoided relationship, and the catastrophe.[6-9]

The group started at a period on the ward when the patients were demanding to have group therapy. In the past, group psychotherapy had been conducted by other therapists (clinical associates or ward administrators) for six months at a time, once a week, and there had been a hiatus of two weeks with no group psychotherapy. At the first session the therapist and nurse-observer entered on time and sat down. The members of the group tried to engage them in diadic communication and were dismayed by the lack of responsiveness. Thereafter, for three months the group was conducted according to a strict Bion model, with only group interpretations made. The group began in basic assumption dependency. Besides attempts at diadic communication, the patients also tried to get the therapist to take over and give advice, or, in other words, take care of them. The interpretation at such time was that the price the group paid for dependency was feelings of frustration and anger. During the first session, when the group desperately tried to get the therapist to take over and treat it as helpless, one of the group members became the repository of the group's dependency and began to hyperventilate. She stated that she was sick and was going to pass out and fell under the therapist's chair. The therapist did not talk or move. The group became furious and one of its members shouted, "This is a nightmare. This is a nightmare. What kind of doctor is this who lets patients die?" The therapist and nurse-observer still did not respond. Finally, when the other members of the group realized that the patient lying on the floor was not going to die, they told her, "Get up and cut the crap." The patient stated that she was very sick and reluctantly returned to her chair. The next session was similar, but this time a highly agitated patient repeatedly walked in and out of the session. The therapist commented that this patient represented the ambivalence in the group, to be or not to be there. Thereafter, the group began to take more responsibility for psychotherapy work in the therapy sessions and to depend less for answers, advice, and pity from the therapist. In fact, the group did this more quickly than outpatient groups and process groups conducted by me, and this would seem to be explained by the fact that the patients lived with each other twenty-four hours a day and knew each other better.[10]

When a manic patient entered the group and dominated the group by his angry invectives directed toward the members of the group and his flight of ideas, the group maintained that they could not conduct the session with such behavior, that they were in fact helpless and hopeless, and asked the therapist to assert his authority to control the patient's behavior. The therapist, in turn, stated that the group supported the manic patient's behavior. The group grew angry at the therapist but after several sessions he was able to control the manic patient's behavior in the group, which the nursing staff could not manage without the use of seclusion. The group dealt with this patient by realizing that when it remained quiet the manic patient took over the group but that when the members were actively engaged in discussing their problems or looking at themselves the manic for the most part remained quiet or was useful to them with his comments. At times when this did not work the group learned that by questioning the masculinity of the manic patient he withdrew and became quiet. The group, however, did not always exercise the power they had in dealing with this patient because he expressed for the group what the group refused to express for itself. It was at these times that the therapist interpreted the group's support of what was going on. This particular sequence was repeated with other disturbed patients, wherein the group members initially acted helpless but were eventually able to control disturbed behavior after the therapist repeatedly reminded them that they supported that behavior.

A 54-year-old woman manic patient constantly harangued, annoyed, and disrupted the group with childish behavior. The group insisted that they could not conduct a session because it was impossible to deal with such behavior. Again the therapist reminded them that they supported such behavior in that she was the repository for the group of these feelings. The group finally dealt with the patient by stating that her behavior was crazy and refused to give her cigarettes while this behavior continued. They also found that if they talked about their feelings, thoughts, and problems, the manic patient would not act childishly and try to engage the group in flight. However, if they remained in a dependent state she would act in a childish way. The group began to understand that their not doing psychotherapy work supported the manic patient's antics even though consciously they disavowed any part of these antics, but by being forced to look at how they supported these antics, and to understand that they were not hopeless or helpless nor omnipotent nor a slave to flight but had power in dealing with what occurred in the group they managed to get work done and at the same time help the manic patient and themselves strengthen controls over her and their own behavior.

At this time the group began to develop into an Ezrielian model. This model not only makes group interpretations but also makes individual interpretations and relates them to the group theme. Also, at this time the first nurse-observer left the group and the second nurse-observer entered. She was also the head nurse on the unit. She participated more freely than the first nurse-observer by sharing her feelings about members and about herself. Her role developed from that of observer to that of active participator by expressing feelings elicited within her by the patients. As the group developed it began to take over more responsibility for ward problems. The patients began to ask for and have meetings about problem patients and problems between patients. The group usually would decide on an operant conditioning model of reinforcement. This was interesting in terms of the therapist who believed in and espoused these views at community meetings. Operant conditioning model as used on this ward was the use of positive reinforcement to produce desirable behavior and the use of negative reinforcement for extinguishing undesirable behavior. This entails observing what is and what is not positively and negatively reinforcing for the individual patient. Therefore, in regards to a very regressed patient who would not flush the toilet, keep her room neat, or dress herself the group held a meeting and decided that this patient's roommate, who was a positive reinforcement to the patient, would move out of the room and not return until the room was in order and the patient dressed herself. This behavior, which had been occurring for nine months, was extinguished two days after the roommate moved out. There was also a negative reinforcement for the patient in being told what a slob she was by the members of the group. The positive reinforcement of being complimented on doing a good job and having her roommate back sustained this new behavior.

The patients established the mores that there was a serious commitment to the group and members could not do as they pleased, when they pleased, or how they pleased. When one 37-year-old woman became manic and expressed a great deal of hostility and anger at the group members, the members initially responded in ways which were characteristic. One member who could not express anger at her husband stated that she refused to talk about her anger toward the manic patient. Another patient who repeatedly left her husband for a mental hospital when angry and depressed, moved her seat away from the manic patient to the opposite side of the room. Another patient, the hyperventilator, talked about her difficulties with her sore throat. This was interpreted to the group and to individual members. After the group session the members held their own meeting without the knowledge of the staff and decided that as long as the manic patient continued to act in an angry, vicious fashion, they would ignore her. In subsequent sessions it was interpreted that the group made the manic patient the repository for their angry and hostile feelings and then dealt with this by righteously excluding her and by running away from her and these feelings which resided in them. The manic patient in turn, it was interpreted, allowed the group to make her the repository for these feelings by her behavior. She was labled a "sap." In subsequent manic periods for this patient, although she fought bitterly with the staff, she managed to get along with the patient group.

Observations About the Group

The group required what I shall refer to as a "critical mass" for psychotherapy work to be done. This critical mass is defined as the number of verbally active members necessary for the psychotherapy work to be done. In my experience the group process necessary for psychotherapy work is impeded with less than four and more than seven verbally active members. I see this as analogous to nuclear fission in that, if critical mass is not attained, there is no energy release. If critical mass is reached too quickly or is too great, there is an explosion (atomic bomb) in which the energy released is difficult to harness usefully and causes great energy waste. If the nuclear fission is controlled (nuclear reactor), usable energy is released. Thus, in the group, fewer than four verbally active members tend to stifle the group process although it still exists. Greater than seven verbally active members tend to make the group process cumbersome and unmanageable.

Originally, many members would not interact because of severe depression and a great deal of dependency. If the level of verbally active members fell below the critical mass the group sat quietly. This occurred over a one and a half month period every other session. The frustration of just sitting and not interacting would activate certain members and this would bring the level above critical mass only to fall off again the next session. Toward the end of the year the group had at least five members who could interact at all times, and the critical mass was sustained so that controlled group interaction occurred.

Work was done with both models, the Bion and the Ezrielian, which are basically modifications of one another. Neither model seemed better able to get the work done than the other. The therapist, however, found it easier to work within the Ezrielian model.

The literature and experienced group therapists have been unanimous in the belief that homogeneous depressed groups are all but impossible to run.[11,12] Under the conditions which existed on the unit a depressed group (mania did not last long and was not frequent) was run out of necessity and homogeneous severely depressed patients comprised the group. The group was difficult to work with but not impossible and at times very rewarding for patients and therapist.

One of the advantages of the group being homogeneous was that the members were able to more easily and clearly see themselves or a part of themselves (often a repressed part of themselves) reflected in the interaction of other group members.[13] The group had the experience of seeing the other members reacting in the way they themselves acted or could experience behavior which contrasted with their own, although the former was much more frequent than the latter because of the homogeneous nature of the patients. Thus, one member who had denied her angry and hostile feelings toward her parents and members of the group many times, became in touch with these feelings as a result of seeing other members express anger and also because there was consensual validation by the group that she was angry and hostile much of the time. The members were able to know themselves better by the effect they had upon others and the picture that the other members of the group formed of them.

It was noted that toward the middle of the year there was evidence of a great deal more patient demand and overt competition among patients for staff attention. There was also a great deal more discussion in group about the demands and competition and the feelings engendered. This seemed to be a function of feelings of safety and risk-taking on the part of the patients, developed as a result of trusting each other more.

The history of the group can be traced as an entity unto itself from beginning to end. The gains the group made and the mores established continued, although the membership of the group changed.

The function of a patient in a manic state in the group was always that of leader, either dependent leader, fight-flight leader, or pairing leader. This function was abrogated at the end of a manic episode. The group, especially the manic patient involved, realized the futility of the political atmosphere of "I'll do for you if you'll do for me," because inevitably the manic patient would end up feeling used by the group and the group would end up feeling angry with the manic patient for not being able to accomplish for it what the group could but refused to accomplish for itself.

It was noted that those patients who were most entrenched in their character structure maintained their roles most persistently and tenaciously for the group as compared to those patients in the group who were less entrenched. Thus, throughout the course of the group two of the three patients who had remained for one year (in this case, an indication of the degree of impairment) maintained consistently the role of dependency and the role of fight-flight. The dependency role was reflected by other patients dressing the patient and escorting her to all activities, while the fight-flight role was reflected in the patient consistently fighting with other patients and running off the unit on several occasions when the ward was in turmoil. These roles would be relinquished at times to acutely ill patients but were always resumed at a later date.

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You Don't Have to be Crazy, But it Helps*

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Myth, folklore, old wives' tales—all these die hard. The field of vocational rehabilitation of the hospitalized mental patient is no exception.

Ask almost any mental health worker to describe the patient with the most rehabilitation potential. Chances are the answer will be: young, bright, educated, motivated, and relatively well put-together psychologically. Chances are, also, that when asked to define "put-together psychologically" our representative mental health worker will talk in terms of perceiving situations realistically and responding to them flexibly. When we ask how he knows that patients with these characteristics are more likely to succeed, the answer will be "personal experience," "my own observation," or even "everybody knows that."

A search of the vocational rehabilitation literature for predictors of who will succeed and who will fail is only slightly more helpful. For the most part, the research is in the descriptive, or pre-experimental, stage. Only two factors predict who will be successful rehabilitation candidates with some regularity—sex and chronicity. Repeatedly, men have proved more likely to succeed than women. Patients with short-term illness are better prospects than those with longer-term illness.

Several of the researchers strongly recommend that their successors focus on motivation as the X-factor which really separates the successes from the failures. The research being reported in this paper is aimed at just that, within a framework of accepted psychological theory. The project was designed as a traditional psychological experiment—the patients were tested to measure particular responses, then re-tested to detect possible changes after exposure to controlled experimental procedures (the treatment condition).

Theoretical Psychological Framework

Over the last 15 to 20 years McClelland and a host of co-workers have been investigating a motivational system he calls "need Achievement motivation."[1] Stripped to its bare bones, need Achievement has to do with a person's desire to compete, either with others or with standards he imposes on himself. McClelland traces its origins to the Protestant Ethic, the belief that each individual is accountable before God, that the way to Heaven is through working very hard to achieve excellence here on Earth. Historically, these beliefs are closely associated with the middle class and with the high value which that class places on personal independence, striving, and getting ahead in the world.

McClelland devised a method of measuring need Achievement motivation, using a special scoring system applied to stories told in response to the pictures on Thematic Apperception Test (TAT) cards. Through many research studies with non-hospitalized subjects, many of them middle-class, the picture has gradually come clear of what a person is like who has high need Achievement motivation—he enjoys competition, throwing himself into it enthusiastically. When he does not have another person to compete with he sets a goal for himself (a level of aspiration) and strives for that. Given a choice of a range of tasks, from very easy to very difficult, he settles for something in the middle. In this way he has the best of all possible worlds—the challenge of a task that is not too easy plus a reasonable chance of succeeding. He is realistic in his assessment both of his own abilities and of the situation he faces. If his first approach to a problem does not yield a solution, he is flexible enough to try some other way. Asked what he values most, he stresses personal independence and a feeling that he controls his own destiny.

In contrast, the person who scores low in need Achievement avoids all competition. Rather than choosing a moderate and attainable goal, he either picks something very easy or, at the other extreme, something impossibly difficult. Having assured his own failure by attempting the impossible, he counts on the cheers of all by-standers for the valiant try. He rejects personal independence, feeling that he is a pawn in the hands of fate.

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Parker and Kleiner, in *Mental Illness in the Urban Negro Community,* a study based on interviews with 3,000 Negroes in Philadelphia, found that those who were disturbed set unreasonably low or high goals for themselves as compared to normal subjects.[2] These authors established that one of the causes of mental illness was goal-striving stress, but this stress which mental patients feel is not simply a matter of personal motivation. It occurs in a social setting. Parker and Kleiner investigated the impact of the social setting in terms of social comparison, self-identity, and self-esteem. Their two chief instruments were a structured interview and the Self-Anchored Striving Scale, both of which were adapted for use in the current study.

The Subjects

The subjects of the experiment were all the male referrals to the D.C. Department of Vocational Rehabilitation Unit at Saint Elizabeths Hospital between April 15 and September 1, 1970. Beginning in May all male referrals to the Unit at Washington Veterans' Administration Hospital were added. Only five of the 50 patients in the sample were VA patients.

As a group, the subjects represented a long-term, chronic hospital population. They had been diagnosed as mentally ill for an average of nine years, although the length of the current hospitalization ranged from one month to 47 months. Diagnoses spanned all those in the diagnostic manual. The largest group was 14 chronic undifferentiated schizophrenics.

The Experimental Procedure

Each patient was given a battery of psychological tests and a structured interview, chosen to measure both McClelland's need achievement and factors usually found clustered with it, and the social-psychological variables which Parker and Kleiner found to differentiate the mentally ill from the other groups in their study. The patients told stories twice, when shown two equivalent sets of three Thematic Apperception Test cards—once early in the battery under so-called neutral or unaroused conditions, and a second time after instructions to induce psychological arousal and immediately following two tests which Thiesen and his VA colleagues have shown in their need Achievement studies to cause physiological stress.[3, 4]

The experimenter substituted a level-of-aspiration administration of the Digit Symbol test for the level-of-aspiration test Thiesen had used between two administrations of the Stroop Color-Word Test. The Digit Symbol Test is a coding task, matching the proper sign to a list of random numbers from one to 10. In a level-of-aspiration administration the subject must estimate before each of ten 30-second trials how many he will do. The important thing is not the total score but the pattern of his estimates. Does he consistently under- or overestimate? When he finds he is doing one or the other, is he flexible enough to adjust his estimates to what he can actually do? Does he stubbornly cling to a goal obviously beyond his reach? Does he become quite disorganized? Miles, in a study of what he called need achievement in a mixed male and female group of patients at Fort Logan Mental Health Center found that this test alone differentiated rehabilitation successes from failures.[5] Interestingly, however, and contrary to all theoretical predictions, the successes, rather than being the more realistic and flexible, were those who deviated most from expected "normal" behavior. Both Miles and this investigator attributed the anomaly to the larger percentage of women in his sample. Women are notoriously capricious when it comes to need-achievement. Miles' experiment was included in the interest of a replication with an all-male sample.

The final tests in the battery were Strodtbeck's V-Scale, an eight-item test of middle-class values; the Winne Scale of Neuroticism, 30 items taken from the Minnesota Multiphasic Personality Inventory, to measure anxiety; and the Self-Anchored Striving Scale.[6, 7] This last is a very interesting procedure which Parker and Kleiner adapted from Cantrill. The patient is shown a flight of steps, numbered from one to 10. Pointing to the top step, the examiner says, "This step represents the best possible way of life for you. Think about it, then tell me what it would be like." The same procedure is used for the bottom step and the "worst possible way of life." With the scale anchored at both ends in psychologically equivalent values, numerical values can be assigned and manipulated arithmetically. By asking first about the step the patient is on today, then what step he was on before he got sick we get a number which says whether he is feeling better or worse off, and how much.

The criterion of successful rehabilitation outcome used in the study was that currently used nationally by Vocational Rehabilitation one month in paid, competitive employment. By this standard there were 21 successes, designated as Group I, Full Employment. A second group of seven patients was marginally successful, in that they either were placed in a sheltered type of employment (such as Goodwill Industries) or in a foster home, after a determination that this was the maximum of which they were capable. This is Group II, Sheltered Employment/Foster Care. The two failure groups were separated into those who failed while being evaluated by Vocational Rehabilitation, but before any agency funds were spent on them, and those who failed in training or on the job, after the counselors had spent money on them. These two groups are Group III, Failed Evaluation, consisting of 10 patients, and Group IV, Failed Training/Job, 11 patients.

Seven measures taken from the test material were statistically analyzed simultaneously with a computer program for multiple discriminant analysis. A single discriminant function was found which accounted for 58 per cent of the variance among groups and was significant at the conventional 0.05 probability level. Rather than breaking cleanly into four distinct groups, some rather unexpected clusters developed in this analysis. Group I, Full Employment, while different from Group IV, Failed Training/Job, was indistinguishable on this measure from Group III, Failed Evaluation. Group II, Sheltered Employment/Foster Care, was different from the Full Employment Group but was like the two failure groups. The two failure groups differed significantly from each other.

Need Achievement and Middle-Class Values contributed most heavily to the discriminant function in the positive direction. Surprisingly, both Education and "realistic flexibility" on the Digit Symbol contributed in the negative direction. In other words, the successes were less educated, less flexible, and less realistic than the failures.

Despite the very low levels and narrow range of need Achievement found in the mental hospital sample, the Full Employment group ranked the highest in need Achievement, as would be expected from previous studies. The McClelland scoring system for TAT stories proved very sensitive. It is possible to score as high as 33 on a set of three stories. The highest score for any patient was 17, unusually high for this group of patients. The second highest score was only 10.

Although McClelland has found that high need-achievers in the normal population are the most realistic and flexible in their approach to problem-solving in a task situation, the present findings are the direct opposite, for mental patients. On Digit Symbol, the Full Employment group set unrealistic goals for themselves and pursued them rigidly. This surprising and paradoxical finding can perhaps be accounted for by looking at the world through the eyes of a long-term mental patient. From his viewpoint he must choose between a known, comfortable world to which he has long since adjusted and the unknown risks and uncertain rewards outside the hospital gate. Perhaps if he were realistic he would choose either not to make the attempt or to fail along the way, as the two failure groups do. Rather than doing that, it could be argued that the successes deluded themselves into employment. Although the lowest in education, they appeared to have fallen the least below their parents in social position on the Hollingshead Index.[8] They seemed to believe that this gap was not so great that it could not be closed. Propelled by their need Achievement motivation, overly optimistic about themselves and their chances of success, they blindly pursued their goal in a way that to the outsidor looks unrealistic and inflexible but that for them was their way to success.

Group III, Failed Evaluation, appeared to be as well equipped to succeed as Group I. They were indistinguishable statistically on the discriminant analysis. The difference between the two groups seemed to be that Group III was much more realistic in a number of ways—in their performance in a task situation, in how they felt about themselves, and in their expectations of how the world would treat them. Adding all these realistic expectations together, they settled for the moderate and achievable goal. They stayed in the hospital, not even making a serious attempt to get a job and get out.

Group IV, Failed Training/Job, ranked low in need-Achievement and Middle-Class Values, despite apparently being the children of high need-achievers, many of them middle-class. Their family background, plus their being the most realistic and flexible of the patients in the study, seems to have given them a deceptive appearance of being good rehabilitation risks. But they share with Group III a poor opinion of themselves and a more pessimistic outlook on the world. This seems to be the group Miles referred to as "failing gloriously," expecting applause from onlookers for the valiant try.

Group II, Sheltered Employment/Foster Care, was the lowest in need Achievement and in the discriminant function did not differ significantly from either of the two failure groups. From observation, their being "successes" is probably attributable to an entirely different dimension than that in this study. They are usually likeable people who seem to have a talent for getting others involved with them. Their low need Achievement would indicate that they are often quite passive and can easily be propelled in almost any direction.

Numerous items of biographical and attitudinal information were also collected and analyzed with the traditional success/fail criterion of most previous rehabilitation studies. In contrast with widely held assumptions, the older patients were not less successful, the better educated were significantly less successful; socio-economic status for both patients and their parents was significantly lower for the successes. As would be expected from the need Achievement literature, Protestants were significantly more often successes than those in other religious categories. An unexpected finding was the tendency for those who denied being religious to cluster in Group III, Failed Evaluation.

In the light of the findings in this experiment, what can we say to our mental health worker who is convinced that the patient with the most rehabilitation potential is young,

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bright, educated, motivated and relatively well put-together psychologically? Young? Age didn't make any difference. Bright? IQ didn't make any difference. Educated? Education proved to be a handicap; the successes were less well educated than the failures. Motivated? Yes, those with high need Achievement were more likely to succeed, but this is a tricky area. The patients who failed in evaluation also had high need Achievement; unfortunately, they combined this with being more realistic about themselves, their abilities, and their expectations of how the world would treat them. The successes, the high need-Achievers, were unrealistically optimistic about themselves and their chances of success. They were inflexible in pursuit of their unrealistic goals. So much for being well put-together psychologically. You don't *have* to be crazy, but it helps, if you want to get a job and get out of a mental hospital.

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A Pastoral Ministry in a Mental Hospital

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Misconceptions about the work of a clergyman are common in all walks of life and this is equally true in the hospital situation. Many of the hospital's staff have little understanding as to how the chaplain functions in the mental hospital setting. This paper is presented to show how the chaplain with his training and expertise in religion can contribute to the welfare of patients and how he can work effectively as a member of the professional hospital staff. It also points to the functions of the chaplain as a vital link between the institution and the larger community.

Among mental patients there is almost always the lack of a sense of meaning and purpose coupled with a low self-esteem. Religion through the community of faith offers a means of restoring a sense of self-worth. The chaplain in his ministry points to eternal dimensions of life that can bring hope to those who have little or no hope.

Religion traditionally deals with man's attitudes toward self, others, and God. These attitudes can be positive and life-affirming or they can be negative and life-defeating. Because of his special training and skills, the chaplain is sensitive to the helping or hindering aspects of religion. Religious resources can be used to assist the patient in developing positive, life-affirming attitudes. The pastoral ministry to a patient may include worship, prayer, sacraments, scripture, hymns, pastoral visits, and counselling. Through these and other resources the chaplain hopes to make the patient more aware of the enabling power of a loving God.

There is no correct model for a pastoral ministry to the hospital community. The style of a pastoral ministry will vary according to the chaplain and the religious background and experience of the patient receiving the ministry. The following illustrations describe certain aspects of a pastoral ministry to patients and also a pastoral consultation with members of the professional staff. They are not intended to demonstrate integrated therapeutic results. They demonstrate how religious resources can assist patients and staff in facing existential anxieties, in meeting dependency needs, and in coping with the need for community.

Certain aspects of a pastoral ministry in "R" Building of Saint Elizabeths Hospital are described by the following illustrations. The patients assigned to this division of the Medical and Surgical unit are either chronically ill or physically incapacitated. They are mostly regressed, in poor contact, and generally receive little more than custodial care. Prior to the institution of daily ward meetings in June 1971, most of the patients demonstrated lack of interest in any interpersonal communication. The stated goal of the daily ward meetings was to help the patients live more self-fulfilling lives on the wards.

The first incident describes how a pastoral ministry includes confrontation and interpretation in dealing with existential or normal anxieties associated with death and separation. The resistance to a discussion of death and dying by some members of hospital administration and staff has been the subject of many recent articles. This resistance was evident in an incident that took place both in a ward meeting and in a post-evaluative staff session.

A patient had died on the ward the previous evening, and all who were present at the ward meeting were keenly aware of his absence. One of the patients expressed the feeling that he was lonely because he missed the one who had died. This stimulated some disconnected discussion by staff and patients about death and dying. It was the first emotionally-charged issue that had arisen since the ward meetings were started, but the subject was quickly dropped.

This incident was later discussed in the staff evaluation session, and the question was raised as to why this kind of discussion was discouraged. Some staff members thought that a discussion of death upsets the patients and should be discouraged. There were comments on the concomitant feelings of fear, guilt and loneliness that had been evident during the ward meeting.

Based on some years of experience in ministering to dying patients, the chaplain made some interpretative comments on the emotions associated with the dynamics of grief and the fear of dying. The importance of facing and discussing openly some of the fears associated with this phenomenon was stressed. The statement was made

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that often staff members were more upset by a discussion of death than the patients. Several of the staff members indicated that this discussion was helpful to them. They spoke freely of how they became attached to patients and grieved when they died. By facing more realistically their own feelings, staff members could help the patients face their mixed emotions about death.

At a subsequent ward meeting following another death on the ward there was discussion of the death and accompanying feelings. Members of the staff helped the patients express their fears concerning death. The fact that both staff and patients were able to discuss more freely their feelings about death was an indication that some openness had been reached. They were also able to articulate some of their anxieties surrounding separation.

The news of the impending closing of this unit of the Hospital stirred up similar anxiety feelings among the patients and staff. In a Holy Communion service the gospel account of the institution of the Lord's Supper was read. The anxiety of the disciples over their separation from a beloved friend and his impending death was discussed. His words of assurance were stressed. It was pointed out that we can know God's abiding presence in times of separation and thus have a sense of hope in times of anxiety, loneliness and death. Subsequent statements from staff and patients indicated that this had been supportive in preparing patients for the proposed change. It was recognized that separation anxieties are a component of feelings relative to death. An experience of trust gives a person the capacity to cope with his separation anxieties and also his feelings concerning death.

The second incident illustrates how a ministry of worship and hymns can assist a clinging, dependent patient find meaning in her life. It also demonstrates how a chaplain is often able to work constructively in co-operation with other staff members in meeting the dependency needs of certain patients through the use of religious resources. A 47-year-old woman with a congenital spine defect had been spending most of her time in a wheel chair. She kept her head bent down almost touching her abdomen. With considerable effort she could raise her head and talk. Most of the time, however, she refused to do this. She was very dependent, demanding, and self-centered. Pastoral conversations with her revealed she had been taken to church regularly throughout her life prior to admission to the hospital. Church hymns had been an important part of her life. Although illiterate, she had memorized certain meaningful hymns of worship and inspiration. She was encouraged to raise her head and sing during the worship services. She did this, and with the encouragement of the chaplain and other staff members she was able to sing a solo hymn for the congregation. She led the other patients in singing some unfamiliar hymns. She was able to contribute something of herself in this act of worship. She looked forward to a service of worship as the most meaningful event of the week.

On one occasion this patient said she would have been a missionary had she not been born crippled. It was explained to her that a missionary was a person who had found meaning in life by helping others. She could also find a sense of "mission" on the ward by being helpful to other patients and members of the staff. Then she began to visit other lonely patients. She encouraged others to verbally participate in the daily ward meetings. She still needed encouragement and support from the chaplain and other members of the staff, but she was making progress in focusing some of her time and energy on others. In this way she was finding new meaning to her life on the hospital ward.

A third incident illustrates a pastoral ministry to a noncommunicative patient through Scripture and Holy Communion. It also demonstrates the importance of community for the general well-being of patients and staff. A 37-year-old man had been a patient at St. Elizabeths since he was 14 years old. His condition continued to deteriorate both physically and mentally. From 1959 until very recently he consistently refused to speak above a whisper. Recently he has participated regularly in the worship services and has learned to repeat the 23rd Psalm and the Lord's Prayer. He has thus been able to verbalize with other patients an affirmation of God's concern and protection. He now speaks his name aloud and will respond to questions. His regular participation in the worship services has contributed to this. As he became more aware of others he gained courage to affirm his faith in God and in himself.

A significant event took place on the ward when this patient and his mother participated jointly in Holy Communion. The mother told the chaplain this was one of the happiest moments in her life. This particular Holy Communion service was a dramatic illustration of a community in fellowship with God and with one another. Patients and staff became more keenly aware that the hospital community does not exist in isolation from the larger community. It is a community within a larger community and needs to consider patients in relationship to their families. The chaplain can and does at times function as a bridge-builder to the larger community as he ministers to the families of patients. Because of his many consultative relations with pastors in the commuity the chaplain functions as a valuable link between the institution and the community. In his speaking engagements and consultations in churches, the chaplain has a unique opportunity to stress the interdependence of hospital and community. He also is able to assist pastors to better use religious resources to promote mental health. He presents the challenge to the community that life is filled with life-giving resources that promote healing.

Needless to say, the pastoral ministry of the chaplain is not accepted by every patient. Some have been deeply hurt by unpleasant experiences with the church or representatives of religion. Some cannot accept the responsibility that is concomitant with the receiving of love. Others can only be reached by a different type of pastoral ministry. The chaplain learns to be flexible in his approach to those from different religious backgrounds and experiences.

The Drug-Free Therapeutic Community at Saint Elizabeths provides an illustration of another style of pastoral ministry. The patients in this area are former drug addicts who live together in the drug-free rehabilitative community. They range in age from 16 to 30 years. They illustrate in a dramatic way the "Mad at God syndrome" referred to by Dr. Stamey[1] in an article by that name. These patients have a great deal of hostility that they have previously "acted out" in anti-social behavior. They are now in the process of learning to cope with their anger in a constructive way.

Religious interviews with those living in the community revealed that 18 out of the 21 Protestant patients had strong feelings of resentment toward religion and the church. They spoke freely of being forced to attend church when they were young. When they grew old enough to resist the pressure of their parents or parent surrogates they openly rejected religion and all that it represented. Many have expressed strong negative feelings toward the chaplain because he represents traditional religion.

The chaplain was challenged to meet the needs of these individuals. There have been individual religious interviews, participation in encounter groups, informal discussions and seminars. There has been a willingness to discuss religious issues as they have arisen. Some misinformation and distorted ideas about religion have been corrected. One seminar conducted by the chaplain dealt specifically with healthy and sick religion as it manifested itself in behavior. The participants spoke freely of their attitudes toward religion and how this related to their anti-social behavior. Some began to realize the possibilities of the use of religious resources to help them live integrated and more self-fulfilling lives.

Patients have been encouraged to speak honestly about their negative feelings and hostility toward religion and the chaplain. An example of confrontation and interpretation in an encounter group involves a 17-year-old patient who is the son of a minister. In a 24-hour group marathon he freely verbalized his hostile feelings toward the chaplain and religion in general. In the process, however, he spent most of the time talking about the cruel treatment he had received from his minister father. When confronted with this he began to recognize that his negative, hostile feelings against religion stemmed mainly from the hostility he felt toward his father. His rebellion prevented him from using his own religious resources and strengths in his rehabilitation. Through this confrontation and interpretation he was able to gain some understanding of his negative feelings. Others in the community could identify with this struggle.

Not enough time has lapsed to evaluate properly the long-range effect of this kind of confrontation, but it does point to future possibilities for a pastoral ministry to drug addicts in the hospital. The patients in this area of the hospital are struggling with their dependency needs. They have renounced their dependency on drugs and are seeking a way to meet these needs in a wholesome, life-affirming way. A healthy religious life will do much to contribute to this end.

The total experience of the ministry involves working with groups of people with diverse interests. A religious congregation is not a homogeneous entity. The experiences and training of the chaplain equip him to work in a unique way in a heterogenous, therapeutic community. His training enables him to take effective leadership in the community and show flexibility as he functions as a member of the professional hospital staff.

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The Medical Society of Saint Elizabeths Hospital Historical Notes: 1937–1967

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In 1937 there was not just a change in Superintendents of Saint Elizabeths Hospital but also an *aggiornamento*—a change in the relationship of the medical staff to the superintendency. When Dr. William A. White died on March 7, 1937 he had served for 34 years. As his health declined, and with so much knowledge and experience as everyone's senior, he had hardly shown the need toward the end of his tenure for "input" from his junior medical colleagues. Whoever the new superintendent would be from outside the hospital, however, the medical staff would serve to familiarize him with the hospital and its workings. During an interregnum of six months duration Dr. Roscoe W. Hall served as Acting Superintendent while a government search committee settled on Dr. Winfred Overholser who became the new Superintendent in October, 1937.

The wish for the medical staff to band together in the best interests of the hospital coalesced in the leadership of Drs. Addison Duval, W. W. Eldridge, and Jay L. Hoffman who called a meeting in late spring, 1937 in the A Building Conference Room of the hospital to begin the process of writing a constitution and electing officers for a new society. There was a division of opinion as to how actively advisory, or separate and independent the new society was to be (oral communication—Dr. Duval). An original long-hand written version of the Purpose of the Society read as follows:

The society shall formulate and present advice, on occasion, to the Superintendent of the Hospital in problems affecting the preservation of the high place which has been established for Saint Elizabeths Hospital among the psychiatric institutions of the world; and it shall strive to encourage and stimulate any efforts seeking to advance the present high position of the hospital.

However, at a meeting on July 8, 1937 the advisory role statement was deleted, and in the typewritten 1937 Constitution, attested to by J. L. Hoffman as a true copy, Article II Purpose stated:

The purpose of the Society shall be the encouragement and initiation of efforts tending to promote the best interests of Saint Elizabeths Hospital and the promotion of cordial social and professional relations among its members, both during and after the completion of their active service at the hospital.

The first officers of the Medical Society of Saint Elizabeths Hospital were Drs. A. M. Duval, President; J. L. Gilbert, Vice-President; and J. L. Hoffman, Secretary-Treasurer. Although there had been smaller special-interest societies at the hospital in the past, e.g., psychoanalytic, this was the first large eclectic medical society to be formed.[1]

To launch their new society in a fitting fashion it was decided to hold a "First Annual Banquet." The printed program for this social event, which was held at the Kennedy-Warren on April 14, 1938, listed 120 members by name. Guests of honor for the evening were the newly-appointed Superintendent and his wife, Dr. and Mrs. Winfred Overholser. Dr. Cloyd Heck Marvin, President of George Washington University, was guest speaker. Not shown on the program was the surprise visit of a "famous Viennese neuropsychiatrist" who spoke of his new cure for syphilis by freezing and breaking the spirochetes, etc. This turned out to be a spoof by "Tiny" Ziman, a colleague with talent in composing limericks, playing the piano, and impersonations (oral communications Doctors Kleinerman and Lebensohn).

To put the formation of this new society in 1937 in perspective it should be noted that the American Board of Psychiatry and Neurology had been incorporated in 1934 giving formal definition to psychiatry as a specialty. In American Psychiatry as a Specialty, Bunker[2] stated: ". . . particularly from 1934 onward there took place a progressive and distinct improvement in the status of psychiatry as a medical specialty as measured, that is, by the position and importance given to it in the medical school curriculum." Also, there was an improvement in the status of the psychiatrist's patients in 1938 when mandatory jury trials for commitment of patients to Saint Elizabeths Hos-

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pital were finally abolished and psychiatrists were able to serve in a more humane way on a newly-created Commission on Mental Health.

The Washington-Baltimore Psychoanalytic Society had been founded in 1930 at a meeting in the private office of Dr. Ernest E. Hadley, formerly on the Saint Elizabeths Hospital staff, along with some other then current or former staff physicians. Three years later this group organized the Washington-Baltimore Psychoanalytic Institute.[1] Thus, the Washington area had become one of the major areas for undertaking psychoanalytic training. This benefited Saint Elizabeths Hospital in attracting young physicians to Washington who began to pass up the more remote mental hospitals as places for training.

Dr. William A. White had maintained an eager interest in and active support of psychoanalysis. His awareness of the importance of psychotherapy in the hospital setting led to his assignment, as early as 1917, of Dr. Edward Kempf as clinical psychiatrist, free of administrative duties. As Dr. Overholser[3] put it: "At the time of his death in 1937 Dr. White was, in my opinion, the most eloquent, forceful, and progressive psychiatrist in this country, and he had achieved for Saint Elizabeths, always a highly regarded institution, an enviable position in the ranks of mental hospitals. The esteem in which he was held was indicated by the fact that during his lifetime, largely through the efforts of Dr. Harry Stack Sullivan, the William Alanson White Psychiatric Foundation was established, an organization which has done valuable work in the fields of education and publication."

At the second Annual Meeting of the Society on April 14, 1939 Dr. Hadley was listed on the program as Director of the newly-established Washington School of Psychiatry and spoke on "Psychoanalysis and Research." Dr. Sullivan, listed as President, William A. White Foundation, spoke on "Some Notes on the Therapeutic Interview." Among the seven presenting papers there was also Dr. Walter Freeman, Professor of Neurology, George Washington University, who spoke on "Shock Treatment and Prefrontal Lobotomy–A Comparison." Dr. Freeman had served as Director of Laboratories at Saint Elizabeths Hospital from 1924 to 1934. The Society continued its annual one-day meetings each spring during the 1940s with the papers reflecting the World War II psychiatric concerns and, especially, the service of the hospital to the U. S. Navy in caring for 5,600 of its psychiatric casualties. Each year Dr. and Mrs. Overholser invited the members and their guests to luncheon at the Superintendent's Apartment in Center Building. In the evening there would be the annual dinner at a downtown hotel with an after-dinner speaker. The 1943 meeting featured Leo C. Rosten, Ph.D., Office of War

Information, speaking on "The Psychiatrist and Propaganda." The 1945 meeting featured an untitled address by Margaret Mead, Ph.D., noted anthropologist.

Beginning with the ninth Annual Meeting in 1946 the scientific program became a two-day affair, with the Superintendent offering a buffet supper on Friday evening, and his closing remarks ended the Saturday presentations. The meeting concluded on Saturday evening with cocktails and dinner at a hotel. The annual meetings were well attended, affording alumni the opportunity to return each spring to renew old ties and present their latest papers. None of those who were present have forgotten Saturday evening, June 3, 1950, at the Mayflower Hotel. Cocktails were scheduled for 7:30 P.M., dinner at 8 P.M., followed by a speaker, and then dancing. A world famous neurophysiologist, the late Dr. Warren S. McCulloch, had come from Chicago to speak. He launched into a long and detailed dissertation on the neurophysiology of the occipital lobe of the brain. Soon the wives present had left the room, and the members were torn between hearing Dr. McCulloch out and wishing to get on with the dancing to follow. That debacle ended the custom of having an after-dinner speaker.

The post-war arrival of many young physician veterans seeking additional training and experience in psychiatry invigorated the Society. Interest in a fresh attack upon the problem of the treatment of schizophrenia produced a half-day program at the May, 1947 meeting that contained: "Presentation of Three Cases of Schizophrenia Unimproved by Electroshock Now in the Process of Receiving Intensive Psychotherapy at Saint Elizabeths Hospital" –William Granatir, M.D.; Otto A. Will, M.D.; and Robert Morris, M.D. This clinical presentation was followed by Dr. Harry Stack Sullivan's "Comments on the Problems of Investigation, Therapy and Education in Psychiatry and their Relations to Schizophrenia."[4] There was a sense of excitement as to the psychotherapeutic possibilities in schizophrenia stirred by the presence in the Washington area of Drs. Sullivan and Fromm-Reichmann, and talk of direct analysis work by John Rosen. It was a common practice to pick a patient and work intensively with him. This meant returning to the patient's ward after having been transferred to another Service, which was sometimes frowned upon by the Clinical Directors.

Superintendent's Apartment, Center Building, Saint Elizabeths Hospital, 1962.

During 1947-1948 Dr. Winfred Overholser was President of the American Psychiatric Association, a position attained by every Superintendent before him. The Association's Annual Meeting was held in Washington in May, 1948. That year the annual meeting of the Society was shifted to November. There was a paper on "Aggression" by Dr. Karpman, which was discussed by Dr. Granatir who was one of those active in the discussion groups that met to prepare papers on aggression in advance of the International Congress of Mental Health held in London, England in October, 1948, which focussed on that subject. Dr. Overholser was Chairman of the U. S. delegation to that conference and reported on it to the Society at the November, 1948 meeting.

Dr. Karpman's interest in aggression was part of the considerable energy he put into the study of the psychopathology of crime and the criminal. He contributed scores of papers to the medicolegal literature, and in 1923 he was probably the first to use the technique of psychoanalysis in treating the criminally insane.[5] As Chief Psychotherapist at the hospital Dr. Karpman was a legendary figure with his flowing beard, heavy Russian accent, sheafs of papers in hand, and a challenging manner at meetings. One target was Dr. Sullivan, whose credentials in being truly analyzed and trained in psychoanalysis he questioned repeatedly. It was presumed that Karpman's not being included in the formation of the Washington-Baltimore Psychoanalytic Society in 1930 had a lot to do with this critical stance. Dr. Karpman was an officer in the Washington Psychoanalytic Association which had been founded in

1924. Another target was Dr. S. Katzenelbogen, Director of Laboratory and Research, who represented the psychobiology position of Adolf Meyer. Dr. Katzenelbogen would flush and become uncomfortable in the face of Dr. Karpman's heated challenges. At the 1946 meeting his paper was to be followed by Dr. Karpman presenting a paper on psychoanalysis. He anticipated trouble by stating in his paper on "Psychobiological Therapy": "Above all I do not wish to provoke a polemic on the renowned school of thought, if for no other reason than for the sake of preserving my own emotional equilibrium."[6]

Dr. Joseph Abrahams brought to Saint Elizabeths Hospital in 1947 his developing expertise in group work which he used at first in the old fortress-like Howard Hall where the criminally insane were treated. He went on to create a group psychotherapeutic experience on an experimental basis, which took place in a visiting room of the hospital and was composed of adolescent and young women chronically ill with schizophrenia, and their visiting mothers. In this study he demonstrated that the interpersonal process between mother and daughter played a part in the perpetuation of the daughter's illness and documented the usefulness for research and treatment of the analytically oriented group therapeutic process.[7] Dr. Abrahams kept the hospital in the forefront of group work training and served as a consultant for many years. He led a workshop for the Medical Society in 1962 which I will refer to later.

The Society's function as the premiere scientific and social organization of psychiatrists in Washington gave way as the number of psychiatrists in private practice increased rapidly. The need for an area-wide unified society led to a meeting on January 6, 1949 at the old Medical Society library.[8] Dr. Duval once again emerged—as he had with the Medical Society of Saint Elizabeths Hospital in 1938—as the first President of the Washington Psychiatric Society. The larger size and diversity of this new society, which was affiliated with and later became a District Branch of the American Psychiatric Association, led to its dinner dance eclipsing that of the Medical Society of Saint Elizabeths Hospital as the psychiatric social event of Washington. However, a cooperation began in the form of an annual joint scientific meeting, usually in January or February. The annual 2-day Spring Meeting at Saint Elizabeths Hospital continued to be the best forum for presentation and discussion of original papers by colleagues.

Tending to be taken for granted, and as evidence to Northerners that they were now South of the Mason-Dixon Line, was the racial segregation practiced at the hospital which made of the Society a "lily-white" organization. The hospital administration abided by the Act of Congress of March 3, 1855, which established segregation of patients according to race. When the landmark Supreme Court decision of 1954 struck down "separate but equal" laws this situation changed. Dr. Luther D. Robinson, President of the Society, 1965-1966, and Acting Superintendent since December, 1969 was the first Negro physician to enter the psychiatric residency training program, beginning in July, 1954.

The zenith attained by Saint Elizabeths Hospital in celebrating its centennial year in 1955 is reflected in the gold-colored program of the professional meeting on May 5 and 6, which was sponsored jointly by the Society and the specially created Centennial Commission. Contributions were made by members and foundations to defray expenses, which included those of guest speakers from Peru, Spain, Switzerland, England, France and Germany. American participants such as Drs. Overholser, Noyes, Felix, Brosin, Stanton, Glueck, Lebensohn, Lewis, and Gardner Murphy presented papers covering many aspects of psychiatric administration, treatment and research. After the presentations at Hitchcock Hall there was a dinner meeting at the Willard Hotel with Dr. Gregory Zilboorg speaking on "The Unwritten History of an Inspiration." In his eloquent address he stated that the inspired humanism of Dorothea Lynde Dix was the expression of the humanism that characterized American psychiatry from its very origins. Given the ideational content of psychiatry he concluded his address as follows:

"This is why psychiatry has its own martyrology, and why psychiatry is in itself an inspiration as yet not entirely fulfilled. This is why the psychiatric leader of today is not only a psychiatrist, a healer; history still demands of him that he remain a reformer, an active seeker for improvement, a relentless fighter of human prejudice in the hospital, in society, in the courts of justice, in human relations in general. And because of all this the liberating spirit and the humanistic inspiration symbolized by the deeds of a Dorothea Lynde Dix and the monument that is St. Elizabeths Hospital have remained inexhaustible through the decades that have formed a century, and will and must remain inexhaustible for much more than the century to come".[9]

The spotlight was on St. Elizabeths Hospital during the centennial year with long newspaper articles (POST: March 3, 1955; and an editorial later); magazine articles ("Saint Elizabeths—Pace-setter for Mental Hospitals" in HARPER'S, January, 1956); and an historical issue of MENTAL HOSPITALS (Vol. 6, No. 5, May, 1955) dedicated to the 5 hospitals that opened their doors in 1855. Dr. Jay L. Hoffman, President of the Society, 1954-1955, and in charge of the Program Committee, added to the recognition he had earned in being one of the triumvirs who had founded the Society in 1937. Unfortunately, he suffered a heart attack at the dinner meeting of the Society on May 4, 1957 and died that night.

The centennial year construction work on a new admission building was completed with the dedication of the Dorothea Lynde Dix Pavilion by Richard M. Nixon, then Vice-President of the United States, on Friday afternoon, April 13, 1956. This dedication formed part of the Society program and followed a morning of scientific papers. Then, significantly, on April 14 there was a panel discussion entitled, "Re-evaluation of the Concerns and Aims of the Medical Society of Saint Elizabeths Hospital" in which the members reflected Zilboorg's description of psychiatrists as active seekers for improvement. Dr. Duval was moderator, and Drs. Farley and Granatir were discussants. The Society was serving its members by mailings that kept them posted about news and events, by reunions at the annual meetings of the American Psychiatric Association, and by offering a forum for their papers at the Spring meetings. Also, since 1952 the Society offered annual awards for papers by interns and residents in training, known as the William Alanson White Prizes. As a result of the discussion on April 14 it was decided to publish the papers given at the meetings. Dr. Edward I. Kushner became the first editor of the Proceedings and served ably from 1957 through 1964. In addition, individual articles appeared frequently in journals such as the MEDICAL ANNALS.

The Society included many members who had been at the hospital for only a year or two, since their primary interest was in the private practice of psychiatry and psychoanalysis. They served as officers, speakers and discussants at meetings of the Society. A roster of such names would read like a listing of the distinguished psychiatrists actively practicing and teaching in the Washington area today. However, a new emigration now took place, partly because some of the administrators had reached retirement age in federal government service, and others sought new posts elsewhere. Dr. Duval, always a leader, left in 1959 to head up mental health services in Missouri and later in Georgia. Dr. Tartaglino went with him to Missouri, and Dr. Pettit followed in 1960 and then later also went to Georgia. Dr. Cruvant had gone to Missouri in 1953. Interestingly, from Missouri to Washington as a physician-Congressman came Rep. Durward G. Hall (R. Mo.), a member of the Society who sometimes attended one of Dr. Overholser's buffets for the society members. These departures were in addition to the retirement of Dr. Silk in 1953, the death of Dr. Hoffman in 1957, and the departure of Dr. T. C. Fong to Indiana in 1958. The hospital began the 1960s showing the effect of the loss of so many senior administrators.

The changes at Saint Elizabeths Hospital as of 1960 were especially noticeable to me as I began to go there regularly while serving on the Commission on Mental Health, 1960–1963. (I had been there from 1946 to 1948). In 1959 the old Howard Hall for the criminal insane had been closed and the new John Howard Pavilion was opened. With the Dix Pavilion receiving new patients the former WR (Women's Receiving) had been renamed Nichols, and MR (Men's Receiving) had been renamed W. A. White, and was occupied by National Institute of Mental Health research facilities. The medical staff was younger. Tranquilizers were now in extensive use.

The Society changed with the times, increasing its dues from $2 to $3 a year, and re-scheduling its business meetings and election of officers for the fall of the year. The silver-speckled 25th Anniversary Program of the Society, set for April 13 and 14, 1962, announced a Workshop, chaired by Dr. Joseph Abrahams, on "Saint Elizabeths Hospital and Its Changing Relationship to the Community-Professional and Social," in addition to the usual individual papers. A half-day on Friday was spent in a general orientation meeting followed by individual workshop groups. On Saturday afternoon the workshops continued with formulation of reports for a final plenary session. It would be impossible to do justice in this article to this lively and useful workshop which tackled the concern over decreasing participation by members. It drew a large crowd of members who felt revitalized and better equipped to go on by having participated. The 1962 Proceedings carries Dr. Abrahams' report along with a summarizing statement by Dr. Dale Cameron, Assistant Superintendent, and concluding remarks by Dr. Overholser in which he pronounced the Society hale and hearty. He referred to having read about its founding in the Boston papers before he was appointed Superintendent in 1937.

Dr. Overholser was 70 years old on April 21, 1962, which ordinarily is the mandatory retirement age in government service. However, he was permitted to continue in office until October 11, 1962 so as to complete 25 years as Superintendent. He was the last to occupy the 8,000 square feet Superintendent's Apartment in Center Building with its many historic antiques because the General Accounting Office had ruled that fair market value would have to be paid to live there—an impossible rental fee considering a Superintendent's salary (WASH. POST, Oct. 14, 1962). However, the Society has used it for luncheons and meetings under its new name of Staff Lounge. On the afternoon of October 1, 1962 the Society sponsored a farewell reception for Dr. and Mrs. Overholser. They continued to live in Washington and attended meetings until his death on October 6, 1964. The 26th Annual Meeting, April 19 and 20, 1963, had been dedicated to Dr. Overholser on the occasion of his retirement and in

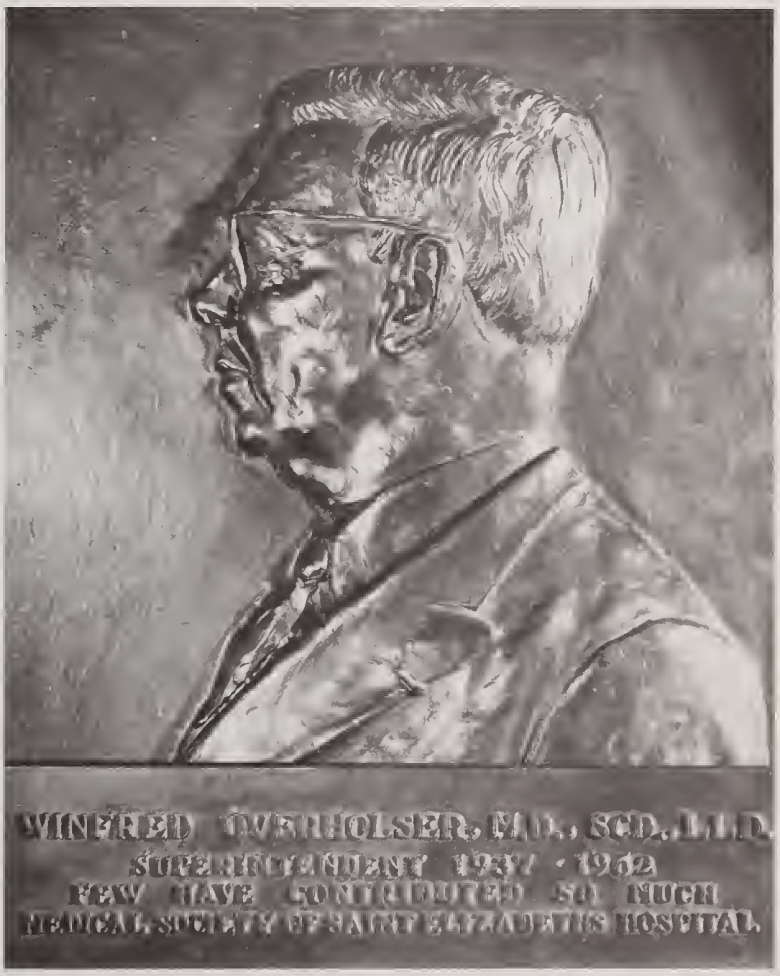
JOHN HOWARD
PAVILION
1959

Winfred Overholser, M.D., Superintendent (left), and Addison M. Duval, M.D., Assistant Superintendent, Saint Elizabeths Hospital, at the John Howard Pavilion setting of the nameplate, March 25, 1959.

memory of Dr. Karpman, who had died on May 17, 1962. It featured "In the Wake of the Durham Decision", a panel discussion which Dr. Overholser moderated and other forensic psychiatry papers appropriate to both Dr. Overholser and Dr. Karpman's interests in the field. The MEDICAL ANNALS of D.C. published the papers from this meeting in July and August, 1963, the latter issue being named The Saint Elizabeths Hospital Number.[10,11] The MEDICAL ANNALS for November, 1965 was named the Winfred Overholser Memorial Number in honor of his service on its Editorial Board. Dr. Zigmond Lebensohn, who succeeded Dr. Overholser on the Editorial Board, furnished an article and secured other suitable papers.[12] On Friday afternoon, April 14, 1967, the Society had an unveiling and presentation ceremony in Hitchcock Hall of a bas relief of Dr. Overholser with a Memorial Address given by Dr. Francis J. Braceland. The bronze profile bas relief executed by Felix de Weldon was paid for by subscription with members contributing willingly. It was accepted by Dr. Cameron on behalf of the hospital and was placed in the professional library. On it is aptly stated, "Few Have Contributed so Much," taken from the text of a statement about Dr. Overholser by his successor as Superintendent, Dr. Dale C. Cameron.[13]

The Spring meetings of the Society continued with the presentation of papers and the awarding of the W. A. White Prizes, the recipients of which since 1952 are listed by Dr. Victor Gregory, Editor, in the 1965 Proceedings. In the 1966 Proceedings Dr. Gregory listed all the officers of the Society since its founding in 1937. Also, the Constitution and By-Laws with changes are printed, and a total of 354 members are reported as belonging.

The 1967 Proceedings, Dr. Robert Robertson, Editor, included the statements made by Dr. Braceland and Dr. Cameron during the presentation ceremony for the bronze bas relief of Dr. Overholser. In addition to the clinical papers, all the Proceedings carry the Superintendent's Reflections, now given by Dr. Cameron who took over from Dr. Overholser in 1962 as the sixth Superintendent of the hospital. In these reflections Dr. Cameron described changing the hospital by creating multiple independent receiving services, thus reducing intra-hospital transfers and providing more continuity of patient care. He noted the Ervin Act of 1964 which provided for the first time for the admission of non-protesting and emergency patients. He noted that voluntary admissions had risen to 39 per cent, reflecting the publicity as to its availability. He attempted to evaluate the likely effects of the newly authorized comprehensive community mental health centers. He gave his "Reflections of Blameworthiness" (1966), in which he expressed some views that had been expressed by me.[10] He related these views to those expressed by the Court of Appeals Judge (now Chief Justice Warren Burger) whom I, as Program Chairman, had arranged to have speak


WINFRED OVERHOLSER, M.D., SCD., LLD.
SUPERINTENDENT 1937 - 1962
FEW HAVE CONTRIBUTED SO MUCH
MEDICAL SOCIETY OF SAINT ELIZABETHS HOSPITAL

Overholser Memorial Plaque.

at a joint meeting of this Society and the Washington Psychiatric Society in January, 1966.

In his April, 1967 Reflections, Dr. Cameron anticipated what was to take place on August 13, 1967, namely, that the 112 year-old Saint Elizabeths Hospital would go under the control of the 21 year-old National Institute of Mental Health. Dr. Cameron resigned in September, 1967 to take an important World Health Organization post in Geneva, Switzerland.

I end this 30-year chronicle at this point, leaving it so some future psychiatrist-historian to continue with the history of the Medical Society of Saint Elizabeths Hospital. That person will find that there are many methodological problems in writing historical notes in the field of psychiatry as described by Mora and others.[14] I will also leave to the next President of the Society a well-made wooden gavel bearing the name of the Society and the inscription, "Presented by W. W. Eldridge, M.D., Pres., 1940" with the knowledge and satisfaction that the Society is still alive and active to the benefit of the hospital with its patients and to the members in 1972.

Acknowledgement:

The author is indebted to Mrs. Josephine D. Skelton for searching for historical materials and photographs at St. Elizabeths Hospital, and to Mrs. Jean Jones, Librarian and Archivist, American Psychiatric Association.

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THE VIEW BOX

(from page 494)

DIAGNOSIS: Arteriovenous Aneurysm or Pulmonary Angioma of the lung.

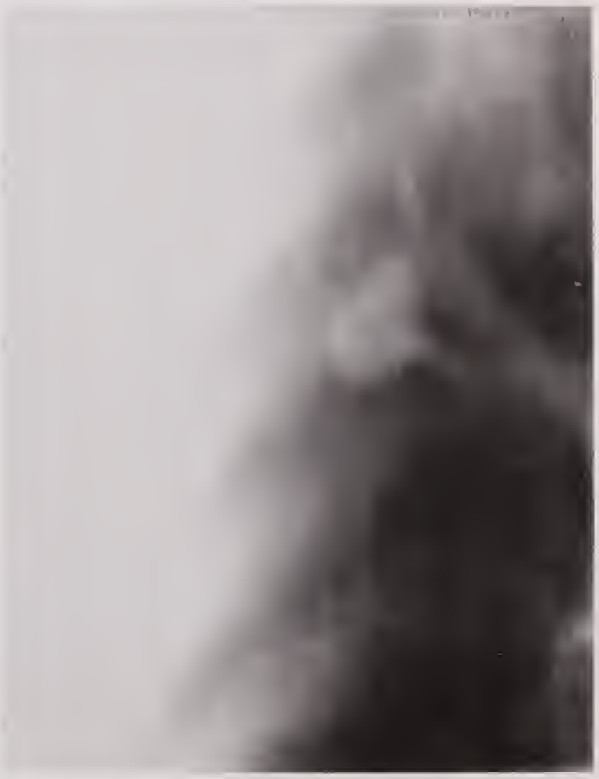

FIG. 2. Tomogram lesion right upper lobe

This consists of dilated "short circuits" between the pulmonary arteries and veins. The clinical picture usually is rather typical, these patients presenting with polycythemia, club fingers and cyanosis. Over the area of involvement in the lungs there are harsh murmurs synchronous with the heart action. Hemoptysis and transient hemeplegias are occasionally observed. Radiographically, the areas of involvement are usually found in the middle and lower lobes and ordinarily vascular strands can be identified extending from the hilus to an irregular nodular area of involvement in the lung parenchyma. With body section radiographs, both afferent and efferent blood vessels from the hilus can be identified. On fluoroscopy these areas pulsate. In the Valsalva test a decrease in the size of the lesion is observed and in the Muller test, an enlargement. Angiography may be necessary to prove the mass is vascular.

Arteriovenous aneurysms of the lung are vascular malformations which in more than 50 per cent of the cases are associated with hereditary hemorrhagic telangiectasis elsewhere in the body (Rendu-Oster-Weber's disease), the patient's chief complaint being that of recurrent gastrointestinal bleeding. The symptoms may become manifest in infancy or as late as the third decade of life.

Edited by Gunter J. Augustin, M.D.
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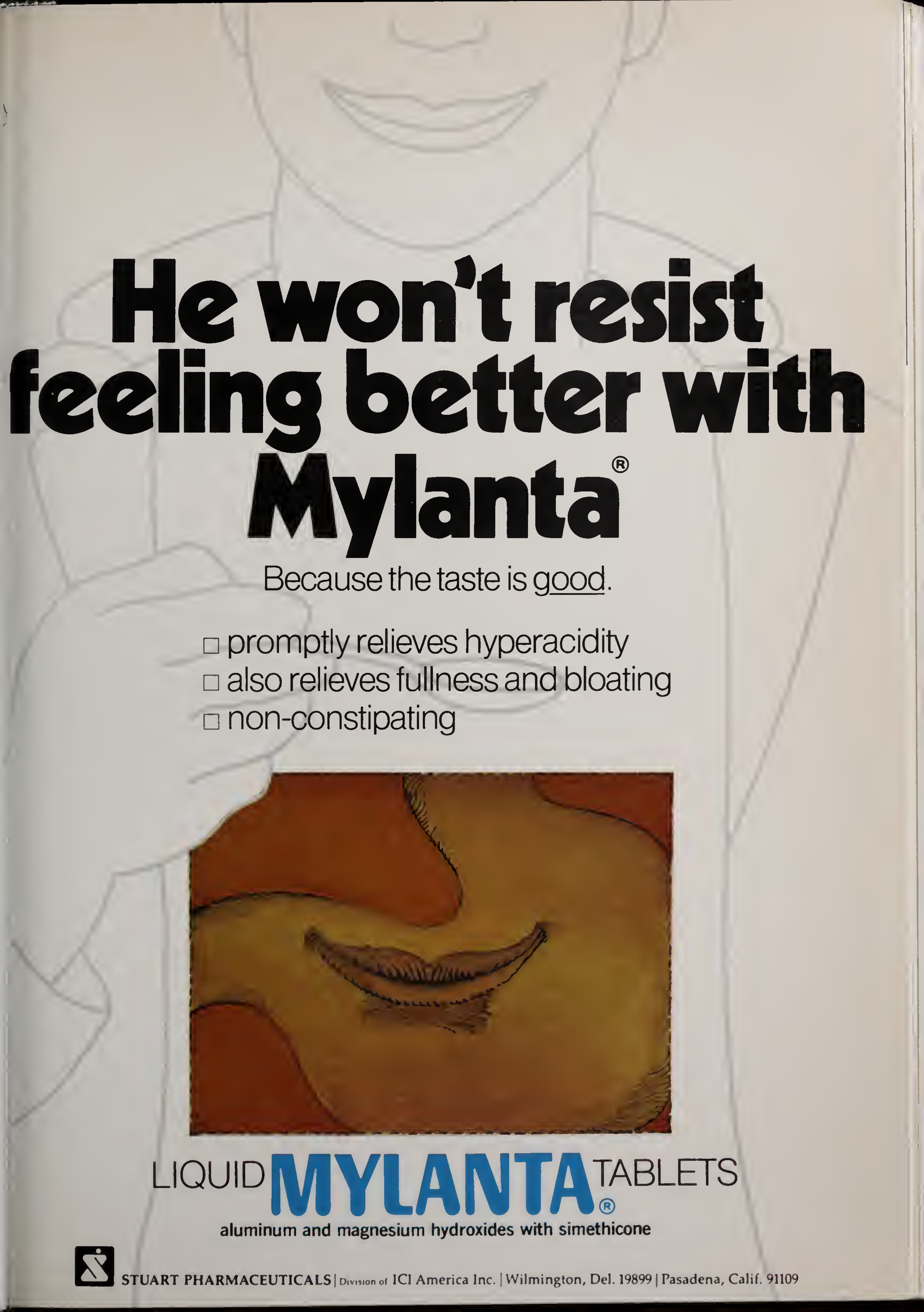
Hospital Center, Washington, D.C.



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If you've seen one, have you really seen them all?

The following patient profiles represent typical clinical situations, but do not necessarily represent actual cases.

Age 21, short, mammose, with normal menses, some acne. Was put on prenuptial regimen of 50-mcg.-estrogen/moderate-progestogen O.C. for two months. Now has increased acne.

Indicates metabolic production of androgen or relative estrogen deficiency.

1st choice: Switch to a 100-mcg.-estrogen combination (such as **Enovid-E*** or a sequential).

Age 25, average frame, poor complexion. No problem with menses, normal para 1. On a low-estrogen/high-progestogen O.C. for two years. Now complains of scanty flow, decreased libido, depression.

Indicates probable buildup of progestogen-related side effects.

1st choice: Switch to a center-spectrum O.C. with more estrogen, less progestational activity (such as **Ovulen***).

Age 19, small breasts, minor hirsutism, oily hair and skin. History of metrorrhagia, skipped or scanty menses. New user.

Indicates androgenic excess or estrogen deficiency (fertility is suspect).

1st choice: An estrogen-dominant O.C. (such as **Enovid-E***).

Age 22, previously normal menses with occasional menorrhagia. Now on a sequential O.C. for four month Complains of heavy flow, occasional intracyclic bleeding, edema, tender swollen breasts.

Indicates estrogen excess.

1st choice: Switch to a combination 50-mcg.-estrogen O.C. (such as **Demulen***).

Unmasked, physiologically and anatomically, they're not all the same. A basic difference lies in their hormone profiles. One may secrete too much estrogen, another not enough…or perhaps too much androgen; the vast majority would fit somewhere into the broad center spectrum.

Although the profiles described below may not be completely predictive, in optimal O.C. selection, the estrogen-progestogen activity ratio should be carefully matched to the patient profile. Searle offers you O.C.s in a range not only suitable for your patients in the balanced center spectrum, but also adaptable to the patient with another type of hormone profile.

Oral contraceptives are complex medications. Among the commonly reported adverse reactions are: intracycle bleeding, fluid retention, tender or swollen breasts, exacerbation of acne condition, changes in libido, amenorrhea while on medication and upon discontinuance, nausea, leg cramps, headaches, weight gain. Therefore, after reference to the prescribing information, oral contraceptives should be prescribed with care.

*Note: In some patients any level of exogenous estrogen or progestogen may produce symptoms of excess hormone activity.

ge 25, tall, slender, athletic,
ith flat chest. On a progestogen-
ominant 50-mcg.-estrogen O.C.
as recurrent trichomoniasis
nd Monilia.
Indicates estrogen deficiency and
xcess of progestogen in current O.C.
1st choice: Switch to a com-
ination pill with 100 mcg.
strogen and less progestational
ctivity (such as **Enovid-E*** or
vulen* or a sequential).

Age 23, "Miss America" figure, previously normal menses, healthy skin and hair. On a 50-mcg.-estrogen pill for four months. Complains of intracyclic bleeding.

Indicates probable need for more estrogen.

1st choice: Switch to a center-spectrum O.C. with more estrogen and moderate progestogen dominance (such as **Ovulen***).

Age 21, college senior, average build. On highly progestogen-dominant/low-dose-estrogen O.C. for six months. Now complains of amenorrhea, between-cycle headaches, weight gain.

Indicates probable progestogen excess.

1st choice: Switch to a center-spectrum pill (such as **Ovulen***).

Age 27, slightly overweight, multiparous. Nausea with all three pregnancies and with a sequential O.C. three years ago. Has premenstrual fluid retention and leg cramps.

Indicates probable excess of estrogen.

1st choice: A 50-mcg.-estrogen/progestogen-dominant pill (such as **Demulen***).

Ovulen® a balanced center-spectrum O.C. for most

Each white tablet contains ethynodiol diacetate 1 mg./mestranol 0.1 mg.

Demulen® a moderately progestogen-dominant O.C. for many

Each white tablet contains ethynodiol diacetate 1 mg./ethinyl estradiol 50 mcg.

Each pink tablet in Ovulen-28® and Demulen®-28 is a placebo, containing no active ingredients.

Both Ovulen and Demulen are available in 21- and 28-pill schedules.

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or a brief summary
f prescribing information,
please see next page.

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Actions—Ovulen and Demulen act to prevent ovulation by inhibiting the output of gonadotropins from the pituitary gland. Ovulen and Demulen depress the output of both the follicle-stimulating hormone (FSH) and the luteinizing hormone (LH).

Special note—Oral contraceptives have been marketed in the United States since 1960. Reported pregnancy rates vary from product to product. The effectiveness of the sequential products appears to be somewhat lower than that of the combination products. Both types provide almost completely effective contraception.

An increased risk of thromboembolic disease associated with the use of hormonal contraceptives has now been shown in studies conducted in both Great Britain and the United States. Other risks, such as those of elevated blood pressure, liver disease and reduced tolerance to carbohydrates, have not been quantitated with precision.

Long-term administration of both natural and synthetic estrogens in subprimate animal species in multiples of the human dose increases the frequency of some animal carcinomas. These data cannot be transposed directly to man. The possible carcinogenicity due to the estrogens can be neither affirmed nor refuted at this time. Close clinical surveillance of all women taking oral contraceptives must be continued.

Indication—Ovulen and Demulen are indicated for oral contraception.

Contraindications—Patients with thrombophlebitis, thromboembolic disorders, cerebral apoplexy or a past history of these conditions, markedly impaired liver function, known or suspected carcinoma of the breast, known or suspected estrogen-dependent neoplasia and undiagnosed abnormal genital bleeding.

Warnings—The physician should be alert to the earliest manifestations of thrombotic disorders (thrombophlebitis, cerebrovascular disorders, pulmonary embolism and retinal thrombosis). Should any of these occur or be suspected the drug should be discontinued immediately.

Retrospective studies of morbidity and mortality conducted in Great Britain and studies of morbidity in the United States have shown a statistically significant association between thrombophlebitis, pulmonary embolism, and cerebral thrombosis and embolism and the use of oral contraceptives. There have been three principal studies in Britain[1-3] leading to this conclusion, and one[4] in this country. The estimate of the relative risk of thromboembolism in the study by Vessey and Doll[3] was about sevenfold, while Sartwell and associates[4] in the United States found a relative risk of 4.4, meaning that the users are several times as likely to undergo thromboembolic disease without evident cause as nonusers. The American study also indicated that the risk did not persist after discontinuation of administration and that it was not enhanced by long-continued administration. The American study was not designed to evaluate a difference between products. However, the study suggested that there might be an increased risk of thromboembolic disease in users of sequential products. This risk cannot be quantitated, and further studies to confirm this finding are desirable.

Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions medication should be withdrawn.

Since the safety of Ovulen and Demulen in pregnancy has not been demonstrated, it is recommended that for any patient who has missed two consecutive periods pregnancy should be ruled out before continuing the contraceptive regimen. If the patient has not adhered to the prescribed schedule the possibility of pregnancy should be considered at the time of the first missed period.

A small fraction of the hormonal agents in oral contraceptives has been identified in the milk of mothers receiving these drugs. The long-range effect to the nursing infant cannot be determined at this time.

Precautions—The pretreatment and periodic physical examinations should include special reference to the breasts and pelvic organs, including a Papanicolaou smear since estrogens have been known to produce tumors, some of them malignant, in five species of subprimate animals. Endocrine and possibly liver function tests may be affected by treatment with Ovulen or Demulen. Therefore, if such tests are abnormal in a patient taking Ovulen or Demulen, it is recommended that they be repeated after the drug has been withdrawn for two months. Under the influence of progestogen-estrogen preparations preexisting uterine fibromyomas may increase in size. Because these agents may cause some degree of fluid retention, conditions which might be influenced by this factor, such as epilepsy, migraine, asthma, cardiac or renal dysfunction, require careful observation. In breakthrough bleeding, and in all cases of irregular bleeding per vaginam, nonfunctional causes should be borne in mind. In undiagnosed bleeding per vaginam adequate diagnostic measures are indicated. Patients with a history of psychic depression should be carefully observed and the drug discontinued if the depression recurs to a serious degree. Any possible influence of prolonged Ovulen or Demulen therapy on pituitary, ovarian, adrenal, hepatic or uterine function awaits further study. A decrease in glucose tolerance has been observed in a significant percentage of patients on oral contraceptives. The mechanism of this decrease is obscure. For this reason, diabetic patients should be carefully observed while receiving Ovulen or Demulen therapy. The age of the patient constitutes no absolute limiting factor, although treatment with Ovulen or Demulen may mask the onset of the climacteric. The pathologist should be advised of Ovulen or Demulen therapy when relevant specimens are submitted. Susceptible women may experience an increase in blood pressure following administration of contraceptive steroids.

Adverse reactions observed in patients receiving oral contraceptives—A statistically significant association has been demonstrated between use of oral contraceptives and the following serious adverse reactions: thrombophlebitis, pulmonary embolism and cerebral thrombosis.

Although available evidence is suggestive of an association, such a relationship has been neither confirmed nor refuted for the following serious adverse reactions: neuro-ocular lesions, e.g., retinal thrombosis and optic neuritis.

The following adverse reactions are known to occur in patients receiving oral contraceptives: nausea, vomiting, gastrointestinal symptoms (such as abdominal cramps and bloating), breakthrough bleeding, spotting, change in menstrual flow, amenorrhea during and after treatment, edema, chloasma or melasma, breast changes (tenderness, enlargement and secretion), change in weight (increase or decrease), changes in cervical erosion and cervical secretions, suppression of lactation when given immediately post partum, cholestatic jaundice, migraine, rash (allergic), rise in blood pressure in susceptible individuals and mental depression.

Although the following adverse reactions have been reported in users of oral contraceptives, an association has been neither confirmed nor refuted: anovulation post treatment, premenstrual-like syndrome, changes in libido, changes in appetite, cystitis-like syndrome, headache, nervousness, dizziness, fatigue, backache, hirsutism, loss of scalp hair, erythema multiforme, erythema nodosum, hemorrhagic eruption and itching.

The following laboratory results may be altered by the use of oral contraceptives: hepatic function: increased sulfobromophthalein retention and other tests; coagulation tests: increase in prothrombin, Factors VII, VIII, IX and X; thyroid function: increase in PBI and butanol extractable protein bound iodine, and decrease in T^3 uptake values; metyrapone test and pregnanediol determination.

References: 1. Royal College of General Practitioners. Oral Contraception and Thrombo-Embolic Disease, J. Coll. Gen. Pract. *13*:267-279 (May) 1967. **2.** Inman, W. H. W., and Vessey, M. P.: Investigation of Deaths from Pulmonary, Coronary, and Cerebral Thrombosis and Embolism in Women of Child-Bearing Age, Brit. Med. J. *2*:193-199 (April 27) 1968. **3.** Vessey, M. P., and Doll, R.: Investigation of Relation Between Use of Oral Contraceptives and Thromboembolic Disease. A Further Report, Brit. Med. J. *2*:651-657 (June 14) 1969. **4.** Sartwell, P. E.; Masi, A. T.; Arthes, F. G., Greene, G. R., and Smith, H. E.: Thromboembolism and Oral Contraceptives. An Epidemiologic Case-Control Study, Amer. J. Epidem. *90*:365-380 (Nov.) 1969.

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Medical Expense Deductions

The Federal income tax laws provide complicated rules that determine how much the individual taxpayer is allowed to deduct for medical care for himself, his spouse and his dependents. "Expenses paid for medical care" are defined to be amounts paid for 1) the diagnosis, cure, mitigation, treatment or prevention of disease, or for the purpose of affecting any structure or function of the body, 2) transportation primarily for and essential to any of the foregoing items, and 3) insurance covering any of the foregoing. The term "dependent" includes a child, a wide variety of other relatives and certain other individuals, but in any case only if over half of the support of such person is furnished by the taxpayer.

Three criteria must be met in order for a child to qualify as a dependent of someone seeking to deduct the child's medical expenses. First, over half of the child's total support must be received from the person seeking the deduction. Second, the child must be a member of the household of the person seeking the deduction; in this regard, a son, daughter, son-in-law, daughter-in-law, stepson or stepdaughter or foster child who lived with the person seeking the deduction during the entire year, except for absences due to school, sickness, vacation and the like, is a member of the household. Third, in general the child must be a citizen or resident of the United States, Canada, Mexico, the Canal Zone or the Republic of Panama.

"Danny Doctor" is in private practice. Whether self-employed or as a partner in a partnership, he has, like any other individual, only a limited ability to utilize the deduction for medical expenses, and then only if he itemizes his deductions. First, Danny can deduct directly only one-half of the cost of medical insurance for his spouse, his dependents and himself, but in any event the amount of the allowable deduction cannot exceed $150. Secondly, he can deduct his uninsured medical expenses only after his remaining medical expenses for himself, his spouse and his dependents exceed the sum of three percent of his adjusted gross income; and only those expenses for medicine and drugs in excess of one percent of his adjusted gross income may be included in this computation. Thus, if Danny has $50,000 in adjusted gross income, and spent $500 for medicine and drugs, no money for medical insurance and $1,500 in other uninsured medical expenses for his spouse, dependents and himself, he would have *no* medical deductions—resulting in an aggregate of $2,000 in expenses without any deduction. Danny would have only $450 in deductions if he spent $500 for medicine and drugs, $300 for insurance for medical expenses and $1,650 in other uninsured medical expenses—an aggregate of $2,450 in uninsured expenses with only $450 in deductions.

From the standpoint of medical expenses and payments, eliminating for purposes of this discussion any other relevant factors, Danny would find it advantageous to incorporate as a Professional Corporation, either individually or along with his partners. In either case the tax saving techniques are available, but they are more easily demonstrated with a corporation in which Danny is the sole employee and sole stockholder. In this regard, it is important to remember that it is equivalent to receiving income on a tax-free basis 1) if Danny does not include a payment to him or on his behalf in computing his income for purposes of the determination of his taxes, and 2) if the Professional Corporation obtains a deduction for such payment.

Assume that the Danny Doctor Professional Corporation adopts a medical reimbursement plan (the "Plan"). The Plan can pay Danny directly, reimburse him or provide insurance for the medical expenses (as previously described) incurred by Danny, his spouse or his dependents. If the Plan is properly adopted, Danny's tax picture under the two examples will be significantly changed. In both of the examples previously described, Danny will effectively be able to obtain a tax deduction equal to the full amount of the medical expenses paid because 1) he does not have to pay Federal income taxes on the medical reimbursement payments made to him or on his behalf by the Professional Corporation, and 2) the Professional Corporation will obtain a deduction for all such payments. The magnitude of this saving is illustrated by the fact that Danny Doctor, if in the 50 per cent tax bracket, could save up to $1,000 in Federal income taxes in the first example by using the technique of the Professional Corporation.

The Professional Corporation can also be used to take advantage of other Federal income tax provisions in the area of health and disability payments. If the Professional Corporation pays money to compensate Danny, his spouse or his dependents for the permanent loss or loss of use of a member or function of the body or for the permanent disfigurement of his spouse, his dependents or himself, none of the money received is subject to Federal income tax, and the Professional Corporation obtains a complete deduction for the payment, as long as the amount of the payment is computed with regard to the nature of the injury and not with regard to the period of Danny's absence from work. Similarly, if the Professional Corporation purchases insurance covering such items, Danny will not have to pay taxes either on the insurance premiums paid by the Professional Corporation or on any payments received from the insurance company, and the Professional Corporation will receive a full deduction for the insurance premiums. If Danny were on his own, he would not obtain a deduction for buying such insurance.

As to other types of disability insurance the premiums for which are paid by the Professional Corporation pursuant to a health and accident plan, although Danny will have to pay taxes on any recovery from the insurance company, Danny still will not have to pay taxes on the insurance premiums paid by the Professional Corporation; and the Professional Corporation still will receive a full deduction for the insurance premiums. For these other types of disability insurance, if Danny were to pay the premiums himself, he would get no deduction for the insurance premiums, but he would pay no taxes on any recovery from the insurance company; thus, by incorporating, Danny gives himself the choice of being able either to pay taxes on the money he uses to buy the insurance and receive any recovery tax free or to have the premiums paid and deducted by the Professional Corporation and have Danny pay taxes on any recovery.

Also, the Professional Corporation provides a vehicle which can be used to prevent taxation on certain amounts paid to Danny under a wage continuation plan as wages or in lieu of wages if Danny is absent from work because of personal injury or sickness. In contrast, if Danny is on his own, he cannot segregate out a certain amount of income at such times and prevent such income from being taxable. In order to take advantage of these provisions, the Professional Corporation must have a plan. After a seven-day waiting period in the event of sickness (but not personal injury), Danny can exclude from income up to a weekly rate of $75 per week for the first 30 calendar days and up to a weekly rate of $100 per week after the first 30 calendar days; there is no such waiting period with respect to personal injury.

One important factor to note as to all of these types of payments is that each of them involves numerous technical requirements and complicated computations. Consequently, even those doctors already operating in the form of a Professional Corporation must take great care to insure that they comply with all of the applicable rules before they can be assured that they will be able to secure the full benefits described in this column.

ANDREW H. LEVY, J.D.

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LETTERS TO THE EDITOR

Minors' Consent to Medical Treatment

June 1, 1972

In "Medico-Legal Report" in the May 1972 issue of the Annals, Mr. Leahy states that the District of Columbia has no statute regarding minors' consent to medical treatment. It is my understanding that D.C. Regulation Number 71-27, adopted last August, gives minors the right to birth control information, services and devices, as well as pre-natal and post-natal care and medical care for their babies if these services are provided by the health facilities operated by the District.

In addition, I understand that the D.C. Code, Section 6-119j-1 allows minors to give consent for their treatment for venereal disease.

Obviously there is need for expanding the regulations to include care provided by hospitals and health facilities not operated by the city government. In the meanwhile, physicians have a resource to which to refer patients who fit the above criteria.

Sincerely,
George J. Cohen, M.D.

Mr. Leahy Replies:

Thank you for forwarding to me the letter of Dr. George J. Cohen referring to a statute and regulation of the District of Columbia involving the consent of minors to the medical treatment described in Dr. Cohen's letter. Unfortunately, as Dr. Cohen points out, the regulation and statute cited by him refer only to minors presenting themselves for treatment to District of Columbia governmental health agencies. The statute and regulation do not provide any criteria under which a private physician may act, except, of course, that a physician may refer minor patients needing the services cited by Dr. Cohen to the appropriate governmental agency. Thus, in the District of Columbia, only governmental health agencies fall under the purview of such statute and regulation, and then only in limited situations. Accordingly, the portion of my article dealing with the District of Columbia remains correct with respect to the treatment of minors by private physicians, and by physicians employed by the District of Columbia, except under the circumstances noted by Dr. Cohen.

Very truly yours,
Michael H. Leahy

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d.c. HEALTH NOTES

Decrease in District of Columbia Birth Rate

Fewer babies are born to District of Columbia residents. The number of babies born to District of Columbia residents declined during the past decade from 19,793 in 1960 to 14,960 in 1970.

The decline in the D. C. birth rate is consistent with the decline in the reported U. S. birth rate. For the United States, the birth rate per 1,000 population was 19.4 for 1965 and 18.2 for 1970. In D. C., the birth rate was 21.9 for 1965 and 19.8 for 1970.

The decline in the number of babies born comes at a time when the use of contraceptive devices and the number of legal abortions have risen, both in the District of Columbia and throughout the country.

In the District of Columbia, the birth control program started in 1964 with a $25,000 appropriation for informational material and supplies. Through the years, this program has grown to include a dozen facilities where women may secure advice on family planning and receive contraceptive devices. All women are examined by a physician, and the types of contraceptive devices are explained to each patient. The "Pill" is selected far more often than any other method of contraception. The cost of this program for 1971 was more than $727,000.

Through the years, the total number of patients served at birth control clinics increased from 1,902 in 1964 to more than 17,000 during 1970 and to more than 18,000 patients during 1971.

When the program started, only married women who had delivered within 90 days and were patients in Health Department facilities were eligible to participate in the program. Eligibility requirements have been relaxed since the start of the program, and today, these services are open to all resident women, whether or not they have ever been pregnant.

The D. C. City Council passed a regulation on August 13, 1971 which stated, "Birth control information services and devices shall be provided by the health facilities operated by the District of Columbia, and may be provided to any qualified person or institution, without regard to the a) age, b) mental status, or c) consent of the patient's parent or guardian."

It should be noted that the fertility rate (number of live births over the estimated total midyear female population age 15-44, multiplied by 1,000) in the District of Columbia has decreased from 97.9 in 1965 to 82.5 in 1970. The decrease is consistent with the fertility rate reported for the United States. The fertility rate for the nation in 1965 was 96.6 and in 1970, the rate was 87.6 per 1,000 women age 15-44.

There has been an out-migration in the District. In 1965, 802,000 residents lived here. During ensuing years, the estimated population decreased, and in 1970, the population was enumerated at 756,510.

Statistical information on population and vital events such as births and deaths for 1969 and earlier years in the District of Columbia are reported in the Vital Statistics Summary. Copies of the Summary may be secured from the D. C. Department of Human Resources, Research and Statistics Division, Room 819, 801 North Capitol Street, Washington, D. C., 20002, or call 629-2697.

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Fay Johnson is an example. Fay was born with no arms, only rudimentary legs and a misshaped head. She had a cleft palate, and her upper lip and lower nostrils never closed. An early check of her reflexes by neurologists revealed that her brain and spinal cord were not developed.

The family went through the usual traumas found in cases of this kind. The father was embarrassed when friends from the office went by the hospital receiving ward to view the child through the window. Unlike most fathers, he didn't stand nearby pointing through the window showing all visitors which child was his. In fact, after his first visit he never went to the hospital again.

The mother was told that the baby couldn't nurse due to a facial deformity, and her pediatrician suggested that she shouldn't be encouraged to hold it until she had better recovered from the effects of childbirth.

Later the mother arrived home in a taxi, without the child. She had been told by the doctors that there was no hope that the child would ever be normal. She and her husband decided against the possible deleterious effects of their three normal children of having Fay in the home, so the child became a ward of the District. Fay's palate was repaired sufficiently so that she could take nourishment, and at about the age of six months, she arrived at Forest Haven.

She is now 16 years old. She eats the equivalent of 16 cans of baby food a day as it is spooned into her mouth by a nurse's aide. Fay stays in bed except for short periods when she is strapped into a seated position in a wheelchair and rolled into the sunlight. These periods must be short because each staff member must take care of many such residents and Fay must be watched constantly when she is out of bed. Even when in bed she must wear a diaper—and now a blouse since she is postpubescent—which must be changed frequently to avoid blistering and bed sores. She makes no intelligible sounds, nor offers any apparent cues to communication.

The point is, Fay has not been put in Forest Haven to die. Dying would be a simple matter for one so helplessly dependent upon the constant watchfulness of others for her mere survival. At one time in the not too distant past it might have been argued that Fay was at Forest Haven merely to survive. We currently feel that Fay has been placed at Forest Haven to live. To make strides toward achieving all the humanity that is within her, this is her birthright.

That may sound difficult in her case, but we feel that it is not impossible. Given the ideal situation we would like to establish a research department at Forest Haven; that is the only way to realistically search out the humanity of Fay and the almost 400 others like her at Forest Haven.

We would like to look into the various kinds of environmental design that would be most suitable for this group. For example, what might be the role of piped in music in their living quarters. Or more precisely, what kinds of music, played at what intensities would be most desirable? What would be its effect on their digestive process? On the level of free-floating stress hormones in their blood streams?

What about use of colored lighting effects to replace the glare of overhead fixtures. Should their open beds be replaced by bunks built into the walls of their living quarters for a greater feeling of security? At what temperature should bath water be?

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Warnings: Use during pregnancy is to be avoided.
Precautions: 1. *Starvation Ketosis:* This must be differentiated from "insulin lack" ketosis and is characterized by ketonuria which, in spite of relatively normal blood and urine sugar, may result from excessive phenformin therapy, excessive insulin reduction, or insufficient carbohydrate intake. Adjust insulin dosage, lower phenformin dosage, or supply carbohydrates to alleviate this state. **Do not give insulin without first checking blood and urine sugar.**
2. *Lactic Acidosis:* This drug is not recommended

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rawn and insulin, when required, and other
orrective measures instituted immediately upon
the appearance of any metabolic acidosis.

3. *Hypoglycemia:* Although hypoglycemic reactions are rare when phenformin is used alone, every precaution should be observed during the dosage adjustment period particularly when insulin or a sulfonylurea has been given in combination with phenformin.

Adverse Reactions: Principally gastrointestinal; unpleasant metallic taste, continuing to anorexia, nausea and, less frequently, vomiting and diarrhea. Reduce dosage at first sign of these symptoms. In case of vomiting, the drug should be immediately withdrawn. Although rare, urticaria has been reported, as have gastrointestinal symptoms such as anorexia, nausea and vomiting following excessive alcohol intake.

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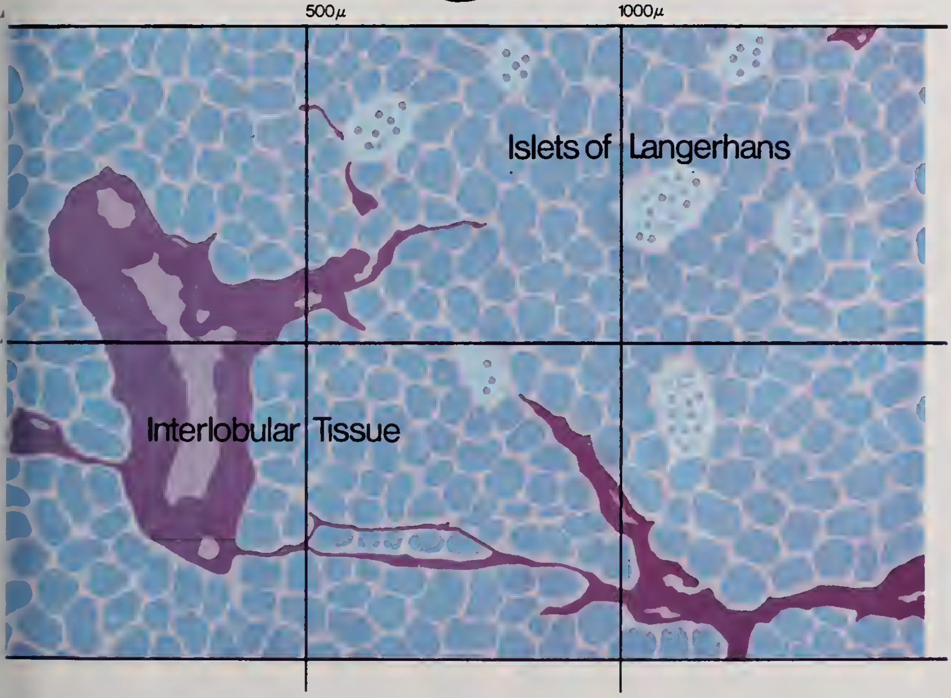
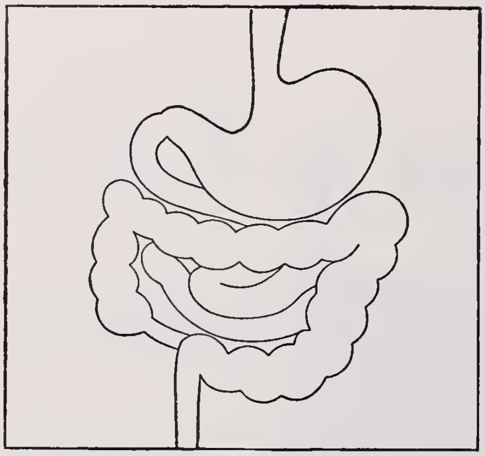


Before using DBI-TD or any oral hypoglycemic, please read the prescribing information concerning dosage, warnings, contraindications, precautions and adverse reactions.

in "Gasspastic" conditions



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cannot meet their basic needs without the frequent or constant help of others, we also have plans. For example, as new buildings are built we would like to continue our current practice of having the heating system radiate from the floors and walls instead of from stoves and radiators as formerly. We would like to have bath water temperature preset so that no resident could scald himself when in the bath or shower alone. In fact, we would like to eliminate all water reaching the residents, above 110 degrees Farenheit or so.

We would like to research new areas of recreation for the severely retarded group, and broaden such programs in both quality and quantity. We would also like to research better and more thorough training techniques, to test the limits of each resident's trainability, no matter what his original level of handicap.

We would like to place at least one severely retarded ambulatory resident in the downtown residences with our less handicapped groups, and teach them to share the responsibility of caring for them as well as for themselves. A good research task.

We would like to research more appropriate clothing for the severely retarded residents. To discover the relative merits of buttons vs. zippers, or perhaps the feasibility of fastening clothes with the new self-sticking cloth tapes only. These could possibly be handled easily by residents who can not hope to cope with buttons.

On and on the list grows. All it takes is money and staff, plus the backing of more of those that cry for change.

For those mothers and fathers who have severely retarded children but who choose to keep them in their home—and there are a surprising number of these families—we would like to offer more services to help lighten their burden and enable them to perhaps live a more normal home life in spite of their handicapped child.

For example, we would like to institute child care activities for such children in the community for those who want to work, or even go on an all day shopping trip now and then, or a two week vacation while we care for the child.

We would like to have a roving teacher to go into the homes and teach the parents basic child training skills geared to the specific handicaps and needs of their child or children. We would like to have workers available to lead discussion groups with such parents, acquainting them with available community resources, encouraging them to share mutual concerns, and even helping them plan new programs for themselves or their retarded children as the need becomes apparent.

In summarizing our Forest Haven reports, we have noted several areas of problems. That is, the things we have mentioned would be called problems by most people; we consider them to be administrative details. They can all be solved or taken care of administratively, and will be. The greater problem is lack of interest in the community, especially on the part of funding agents. In any event, we are no longer willing to see Fay Johnson merely survive at Forest Haven. We are going in search of her humanity, with help or without it.

ESSEX C. NOEL, III, M.D.
Director, D.C. Mental Health Administration

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MEDICAL ANNALS

book REVIEWS

Anatomy for **Surgeons**, Vol. II: The Thorax, Abdomen, and Pelvis, 2nd edition. W. HENRY HOLLINSHEAD, PH.D. Price, $37.50. Pp. 912 with 1102 illustrations. Hagerstown, Maryland: Harper & Row, 1971.

This rather large, wordy volume presents anatomical facts and descriptions of special interest to the surgeon. Complete descriptions of anatomy are not given, emphasis being made on general details of anatomy and physiology relating to surgery. In this new edition, the author states he has made improvements in the interest of clarity and accuracy. The list of surgical consultants who read and criticized the book is impressive.

New material on the lymphatics of the breast and lungs is presented. The section on the blood supply and biliary drainage of the liver, and the segmental structure of the liver is very complete and clear. Of interest are the extrahepatic anastomoses adjacent to the liver and the collateral routes of circulation to the liver which are presented. Considerable space is given to the abdominal wall, posteriorly, laterally and anteriorly, with detailed descriptions of the vessels and nerves involved. A detailed discussion of hiatus hernia and related subjects is especially pertinent in view of the rapidly changing attitudes in the treatment of esophageal reflux.

This text is clearly written and is certainly informative for students, residents and practicing surgeons. Probably in the interest of economy all of the many illustrations are in black and white. But in this day of color, one expects it, even from the moon.

PHILIP A. COX, M.D.

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Neurohypophysial **Hormones**. Ciba Foundation Study Group #39. G. E. W. WOLSTENHOLME AND JOAN BIRCH, editions Price, $8.25. Pp. 146. Edinburgh & London: Churchill Livingstone, 1971.

This 139-page volume consists of nine papers with a discussion following each, in addition to an introduction and a brief concluding statement by Dr. Rudinger, the chairman of the Ciba Foundation Study Group #39 held on 26 March 1971. This was an interdisciplinary group meeting considering the neurohypophysial hormones. The papers and discussions began with a summary of Professor Hans Heller's many significant contributions to this area leading to a succinct but detailed discussion of the evolution of the neurohypophysial peptides in nonmammalial vertebrates by Sawyer. These two early papers were extremely interesting and stimulating to the reviewer. Areas of neuroanatomy and neurophysiology were covered in papers dealing with the mechanisms of secretion of vasoactive materials and the biosynthesis and transport along intraneuronal routes of vasopressin and oxytocin and some of the effects of arginine vasopressin. A short but stimulating paper was that on the present knowledge of the neural hormones which regulate adenohypophysial function by Dr. B. K. Follett and this was followed by a paper by Schild concerning the role of metals and the vasopressin receptors, particularly the relationship of the magnesium ion to vasopressin response. Schild's studies demonstrated that potentiation by magnesium existed for both arginine and lysine vasopressin but that magnesium only slightly and inconsistently potentiated oxytocin's effects. Interestingly, other alkali earth metals, particularly beryllium and calcium, produced either no effect or very minimal effect. The transitional metal group, particularly manganese, cobalt and nickle, were found to be highly active in this potentiating effect.

Other papers also concerned the receptor neural humor interaction at cell sites. The data, although now a year old, I think, will be primarily of interest to investigators in these areas of physiology. The interaction of many authorities from multiple disciplines in the discussions following each paper made for this reviewer extremely interesting reading and offers much worthwhile food for thought, no matter what the discipline of the reader may be.

JOHN J. CANARY, M.D.

New Concepts in Allergy and Clinical Immunology. U. SERAFINI, A. W. FRANKLAND, C. MASALA, AND J. M. JAMAR, Editors. Price, $31.00. Pp. 475 with illustrations. Amsterdam: Excerpta Medica, 1971.

Every three years, allergists and immunologists from all over the world meet in an International Congress of Allergology to review the state of knowledge in various areas of the field and to present new data. This book is the written record of the most recent meeting held in October 1970. Papers range from reviews of topics by leaders in the field, to recent research in specific areas and to abstracts of other areas.

The book is divided into 12 sections, beginning with basic discussions of antibodies and cell-mediated immunity. Bronchial asthma is discussed in three sections: Areas of study and progress, Childhood asthma and Disodium Cromoglycate. Other sections include drug reactions, immune complexes, auto immunity, immunosuppressive therapy, in vitro lymphocyte transformation, animal models of allergic diseases and problems of treatment in allergic diseases. The range of topics goes from the immunochemical and laboratory aspect to the clinical application. The book will appeal to the clinical allergist, clinical immunologist, research immunologist and the many other specialists, whose fields now must include immunology, such as the transplantation surgeon, nephrologist, rheumatologist, etc.

I found the following papers particularly worthwhile. The review of human reaginic antibodies by the Ishizakas is a classic. Dr. Bram Rose's review of the pathophysiology of asthma, Dr. Reed's review of beta-blockade and allergy, and Dr. Pepys' discussion of extrinsic allergic alveolitis provide well-presented surveys. The round table on childhood asthma reviews known information and points to the many unanswered questions. The section on drug reactions presents information on this very complex subject in general terms and focuses on penicillin, ACTH and insulin. Because human allergic diseases are so difficult to study, animal models have served to advance the knowledge in the field and a section reviews these models, including the allergic dog, the only animal other than man who develops inhalant immediate hypersensitivity. The section on immune complexes discusses glomerulonephritis, SLE, rheumatoid arthritis and cryoglobulinemia. Autoimmunity is discussed in relation to gastritis, ulcerative colitis and liver diseases. Dr. Lowell's paper on the evaluation of the effects of drugs provides a thoughtful discussion of the problems of drug evaluation. The section on Disodium Chromoglycate reviews the information available up to 1970. The section on immunosuppressive therapy is excellent.

As would be expected in a book containing papers on such a wide range of topics by workers from all over the world, a few papers do not measure up to the high quality of most of the presentations. A working knowledge of basic immunology would help the reader in understanding the more technical papers. For the physician whose field has been affected by the many advances in immunology of the last two decades, this book is highly recommended.

ISAAC WEISZER, M.D.

Anger. LEO MADOW, M.D. Price, $6.95. Pp. 132. New York: Charles Scribner's Sons, 1972.

This small volume treats anger in the medical mode, it describes the symptoms and signs, traces the normal development and expressions, discusses the pathological manifestations and offers recommendations for management.

The language is clear, simple and didactic and the type, a pleasure to read. The contents are an overview of the frustrations in development and in society with simplified explanations of the dynamics rather than a serious exploration of anger in today's world. The author's preface suggests that the book is directed to unsophisticated laymen who indeed may find it useful. The author's white, male chauvinistic views are apparent thruout and he "explains" that nonwhites and women alike must learn to cooperate with society.

DOROTHY A. STARR, M.D.

medical annals of the district of columbia

vol. 41 no. 8 august 1972

World Trends in Medical Education: Faculty, Students, and Curriculum. ELIZABETH PURCELL, Editor. Price, $10.00 Pp. 238. Baltimore: The Johns Hopkins Press, 1971.

This book is a compilation of the papers presented by leading medical educators from Europe, the United States, Latin America, Japan, India, Africa, the Philippines and Indonesia analyzing the changing trends in medical education in their countries.

The reports are based on the 1969 International Macy Conference on Changing Patterns in Medical Education in the World held at Lake Como, Italy, September, 1969.

Each of the contributors reviews the history of the development of medical education as it is related to the general historical events and the colonial influences that shaped the growth and development of their respective countries.

This is followed by an evaluation of past and present educational objectives, major educational advances, medical admissions procedures, curriculum and graduate training programs. The problems of recruitment of students, applicant pool size, attrition, testing and faculty requirements are also briefly presented. In addition public health problems, "physician drain" and ancillary health science training programs are also included by some authors. A few insights are given into the problem of student unrest and its influences on medical education.

The final transcripts of the panel on the changing characteristics of medical students should be of interest to all physicians whether they are directly involved in medical student teaching or not. The authors try to explain the causes and focus of student unrest and how faculties and administration might deal with these problems. The panels on curriculum, residency training and school governance while not as exciting summarize the basic similarities and differences among the countries represented.

Finally the participants agree that better integration of the scientific and non-specific aspects of the curriculum is essential to provide improved integrated medical care. Liberally dispersed in this amalgamation must be emphasis on social problems, preventative clinical medicine and student involvement at all levels.

An interesting analysis of medical education at an international level. This book belongs on the shelf of all physicians and non-physicians involved in medical education at the faculty and administrative levels.

BRUCE I. SHNIDER, M. D.
Professor of Medicine
Associate Dean
Georgetown University School of Medicine

Human Physiology. ROBERT S. SHEPARD, PH.D. Price, $20.00. Pp. 667 with 585 illustrations plus tables. Philadelphia: J. B. Lippincott, 1971.

With the development of new investigative techniques based on modern technology, the understanding of human physiology is changing and growing rapidly. Nevertheless, some of the basic processes still hold true today as they did a couple of decades ago. The author in his preface clearly states that this volume is for the student—to initiate him into human physiology and he omits information that is well documented in special branches of human physiology, such as nutrition and anatomy of the brain. True to his word the author does present an integrated text on human physiology as an introductory course to the average medical student.

The text is clear, informative and well illustrated with selected figures borrowed extensively from the literature. The bibliography contains references to fairly recent publications. To the curious student these could serve as a source of material for current advances in human physiology.

Although some subjects are covered superficially and do not seem to be of interest to the author, it does contain informative and descriptive diagnostic tables and material which are often hard to locate.

This volume is a worthwhile addition to any medical library. It fulfills the need for a coherent well written general text on human physiology. Students in the biological sciences at the college level, medical students and nurses would welcome such a text book. It also can fill the gap and serve the interest of the practicing physician who wishes to brush up or get re-acquainted with newer aspects of human physiology.

SOLOMON N. ALBERT, M.D.

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Diseases of the Kidney, 2nd Edition. MAURICE B. STRAUSS, M.D. AND LOUIS G. WELT, M.D., Editors. Price, $50.00. Pp. 1456 with illustrations. Boston: Little, Brown & Co., 1972.

When the first edition of *Diseases of the Kidney* was published in 1963 it quickly became recognized as a standard against which to measure other texts. For completeness and readability the second edition maintains the tradition of the first. A few new chapters have been added, and that on Pediatric Nephrology and the two on transplantation are particularly valuable additions. Other new chapters are those on "Hematologic Aspects of Renal Failure," "Calcium and Phosphous Metabolism in Renal Failure" and "Neurologic Complications of Uremia." As in any multi-author text, not all of the chapters are equal in style and content, but nearly all present the historical development of the subject and the pathophysiology as well as the clinical findings and pathology. Although the space devoted to treatment varies considerably in other sections, the chapters "Consequences of Renal Failure and Their Management," "Pediatric Nephrology" and "Clinical Examination of Renal Function" are replete with practical details and provide useful guidelines for patient management for the physician with limited experience with renal disease.

One note of criticism is applicable to nearly all texts of this type, namely that the time required for publication precludes inclusion of the most current work. Even though nearly every chapter has been revised, many of the revisions are minor and do not succeed in bringing the discussion really up to date. It is a little disheartening to observe that a "recent" review refers to a publication which appeared in 1968 or earlier. With rare exception (one reference to a 1970 publication was noted in skimming the bibliographies) the latest journal references are those published in 1969. Although the bulk of the material is nonetheless accurate, some important recent developments are not mentioned. To cite only a few examples, the discussion of "third factor" is rather abbreviated and the role of parathormone in renal acid-base balance is not brought out.

This edition, with is in two volumes, contains a table of contents and an index in each volume, a feature which this reviewer greatly appreciates.

Diseases of the Kidney is recommended as a fine reference work for clinical nephrology. It is particularly recommended for those just entering the field or those who encounter renal disease frequently in practice but are not full-time nephrologists. The reader should always bear in mind that the material being presented serves as a base but that a review of recent periodicals is necessary for more current information.

GILBERT M. EISNER, M.D.

Without Prescription. ERWIN DI CYAN, PH.D. AND LAWRENCE HESSMAN, M.D. Price, $7.95. Pp. 321. New York: Simon & Schuster, 1972.

Ask any man or woman what they most fervantly wish in life and they are sure to include "good health" high on their list. "Health" is big business. No wonder then that the public has been exploited from ancient times to the present with just about every device and drug the human mind could conceive.

Current "over the counter" remedies are the survivors of a vast array of medicaments offered over the millenia of human experience to relieve suffering. They usually do little good and little harm when taken with a large dose of skepticism, a sense of humor about the foibles of man and responsible governmental control. This book takes them seriously and therein lies its strength and weaknesses. It is combination of a home health advisor, compendium of popular over-the-counter remedies and apologist for the industry.

Some of the information it presents is useful. For example clear warnings are given against the use of aspirin in patients on anticoagulants and the potential for aspirin to produce gastrointestinal hemorrhage. Rubefacients should not be used when there is severely restricted circulation in the legs; cod liver oil does not cure hemorrhoids; rectal bleeding occurring in patients past 40 requires a proctoscopy and barium enema; no attempt should be made to treat chronic diarrhea with over-the-counter medications; thyroid preparations should not be used in the treatment of obesity; and other goodies.

Many of the things the authors have to say are silly or gratuitous. For example in presenting an apologia for tranquilizers the authors state that "Society keys up the individual to strive in his competitive endeavor. It should not cast a social frown upon drugs that can replenish his reserves." No mention is made of drug dependence or abuse. Another quote, "One of the purposes of this book is to help the reader measure the price so that he can compare it with the value . . . ," but in no place in this book are monetary prices mentioned. This subject is completely ignored! The authors tell us that women complain of headache more commonly than men therefore suggesting that emotional volatility is a cause of headache. In discussing anal disorders they come out with this deep thought, "there may be penis envy, but no anus envy—." Towards the end of the book they delve into amateur psychiatry and are way over their heads.

Although much useful information is provided on simple pharmacology and the active ingredients of common remedies, the underlying theme of the book is the pretention that over-the-counter remedies should be taken seriously as valuable medications. They shouldn't be meddled with by regulatory agencies, nor be subjected to tests for efficacy even though for the most part they are irrational mixtures concocted to exploit a gullible public. One almost has the feeling that this book was sponsored by the industry to lend respectability to their enterprise. The only item attacked with vigor are feminine hygiene sprays, but even here the authors recommend that they be chosen according to which gives the most pleasant odor.

To sum up, the reader now has much knowledge about many unimportant agents glossed over with minimal guidelines. Nevertheless, the physician should be familiar with these preparations and do something the authors refuse to do—advise their patients on a small number of low cost specific products that may provide relief for his aches and pains, insomnia, constipation and itch. If the physician fails to do this, others are more than willing to assume the role for a premium price.

CALVIN M. KUNIN, M.D.
University of Wisconsin Professor of Medicine

Principles of Child Psychotherapy. DONALD J. CAREK, M.D. Price, $10.75. Pp. 225. Springfield, Illinois: Charles C Thomas, 1972.

Dr. Carek's book "Principles of Child Psychotherapy" is an excellent text for a limited audience. His audience, fellows in child psychiatry and residents in psychiatry participating in training in child psychiatry, are in need of a book on the philosophy of how to practice child psychotherapy. Dr. Carek's book is a philosophy of child psychotherapy. It contains no original theories or treatment plans. Instead, it is an eclectic view with a slight psychoanalytic bias of psychotherapy with children. Dr. Carek presents his material adroitly. His text is not a cookbook on how to do therapy but instead is a very broad and inclusive consideration of the possible moves and options in a therapeutic setting. The possible therapeutic moves and options are thoroughly discussed and the author's implicit hope is that his readers will learn to practice therapy in a rational common sense approach. Above all, Dr. Carek makes a plea for the flexible application of treatment plans. He hopes his readers will learn to fit treatment plans to patients rather than fit patients to treatment plans.

Principles of Child Psychotherapy is a thorough book. It begins with a consideration of the place of child therapy in the overall picture of psychotherapies. In subsequent chapters the diagnostic evaluation, the meaning of play therapy, the basic moves in therapy from beginning to termination, are discussed. The role of the family in child therapy and therapeutic milieu-foster homes, residential treatment centers etc., two subjects most bothersome to psychotherapists in training, are thoroughly discussed with clarity.

SYDNEY G. SALUS, M.D.

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Walter Freeman, M.D.
1895–1972

Walter Freeman, who was President of the Medical Society of the District of Columbia in 1949–50, left an indelible impression on the community and his chosen field of neurology and psychiatry. He was an educator, scientist, clinician, author, poet and crusader. He was first Chairman of the Department of Neurology at George Washington University School of Medicine. He was the first to employ insulin shock therapy, metrazol shock therapy and electroshock therapy in Washington. In cooperation with James W. Watts, the neurosurgeon, he introduced Psychosurgery into this country. Freeman was a Founder Member of the American Board of Psychiatry and Neurology, was its Secretary from inception in 1934 for a period of twelve years when he became President. During these formative years his influence played a big role in setting standards of education and quality. He once said, "We are less interested in a candidate's total knowledge than in his competence." During his tenure every applicant came under his scrutiny and many looked on him as the man who accepted or rejected their applications and passed or failed them on examination.

Generations of George Washington University medical students remember him as a teacher who made neurology a living thing. In the May, 1965 issue of the Medical Annals, in an article on the Department of Neurology and Neurological Surgery, Watts wrote: "In 1935 neurology was taught in the second, third and fourth years. The Saturday afternoon Outpatient Clinic was held in Hall A of the Medical School. Attendance was voluntary; no roll was called, no grades given. Attendance averaged 30 to 35, but I have counted as many as 65 to 70. We were still in the midst of a depression. Walter Freeman was a showman, and medical students often brought their 'dates' (it was cheaper than going to the movies). After examining a patient with a stroke or Parkinson's disease, he would march up to the blackboard, pick up a piece of chalk in each hand, draw a coronal section of the brain, using both hands simultaneously, and sketch in the internal capsule and basal ganglia, dotting in the pathologic lesion. The students were fascinated."

Walter Freeman, M.D.

Much of his time in the last twenty years of his life was devoted to a follow-up of lobotomy patients, so that the end results would be available to the world. He criss-crossed the United States in his camper bus equipped with desk, photographic equipment and file cabinets, often going one to two hundred miles out of his way to interview and photograph an old patient. This was his magnificent obsession.

He was a man of many talents and many faces. When his children were young he loaded them in his trailer and headed for the Yellowstone or one of the large National Parks. He was keen about physical exercise, not only as a method of treating his patients but also for his own vigor and sense of well being. He was mountaineering and on the ski slopes until he was well into his sixties.

Few people were indifferent to Walter Freeman. Psychiatrists looked on him as a neurologist, neurologists looked on him as a psychiatrist, and when he personally began to perform Transorbital Lobotomy in 1949, neurosurgeons considered him a maverick. He thrived on controversy. He was never dull and had something stimulating to add to every scientific discussion.

Born of a distinguished family in Philadelphia in 1895, the grandson of William W. Keen, a noted surgeon, Dr. Freeman received his A.B. at Yale in 1916, and his M.D. from the University of Pennsylvania in 1920. In 1929 he obtained an M.S. at Georgetown and a Ph.D. in Pathology at the same university in 1931. He studied abroad in Rome and in Paris and spoke French fluently. From 1924-31 he was Associate in Pathology at Georgetown. From 1924 to 1927 he was Professor of Neuropathology at George Washington when he was appointed Professor of Neurology. In those days one could hold dual appointments in the two medical schools. He also served as Director of the Laboratory at St. Elizabeths Hospital from 1924-33. He was in private practice in Washington from 1926 to 1954 when he moved to California. During these years he was the author of four books and well over two hundred scientific papers. His Neuropathology was published in 1933 and The Psychiatrist in 1968. His two books on Psychosurgery, with Watts as co-author, appeared in 1942 and 1950. He belonged to many medical organizations and was president of several.

He married Marjorie Lorne Franklin in 1924. Mrs. Freeman died several years ago in California. The great tragedy of their married life was the accidental drowning of their son, Keene, in the Yosemite National Park in 1946. After a memorial service held in San Francisco on June 3rd, Dr. Freeman was buried in Merced, California in the family burial plot close to Yosemite.

Surviving are his daughter, Mrs. Donald Cantor of San Francisco, three sons, Dr. Walter Freeman III of Berkeley and Dr. Paul Freeman of San Francisco, California, and Franklin Freeman of Philadelphia, six brothers and sisters, and fourteen grandchildren.

JAMES W. WATTS, M.D.

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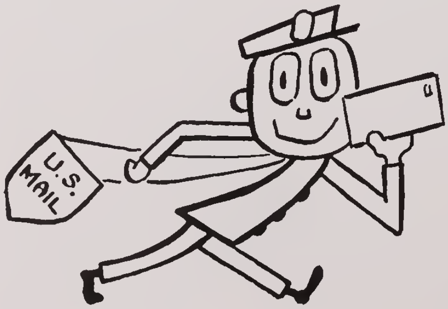

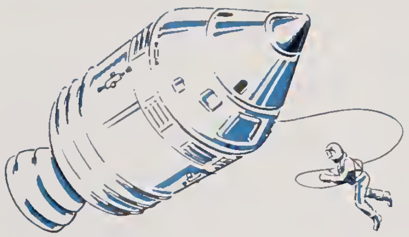

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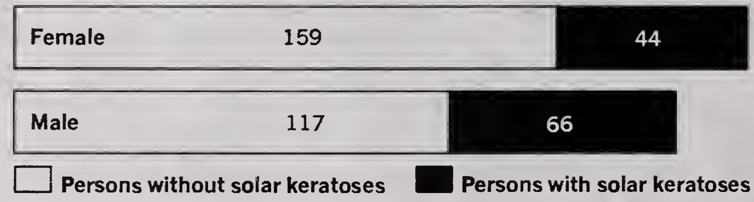
Prevalence of solar keratoses in white persons over 40 in Tipton County, Tennessee



*Data on file, Hoffmann-La Roche Inc., Nutley, New Jersey.

olar, actinic, senile keratoses

lled by many names, the typical lesion is flat
slightly elevated, brownish or reddish in
lor, papular, dry, adherent, rough, sharply
fined; usually multiple lesions, chiefly on
posed portions of the skin.

equence/selectivity of response

ythema in areas of lesions may begin after
veral days of therapy; height of reaction
nly in affected areas)* usually occurs within
o weeks, declining after discontinuation of
rapy. Since this response is so predictable,
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Precautions: If applied with fingers, wash hands immediately. Apply with care near eyes, nose and mouth. Lesions failing to respond or recurring should be biopsied.
Adverse Reactions: Local—pain, pruritus, hyperpigmentation and burning at application site most frequent; also dermatitis, scarring, soreness and tenderness. Also reported—insomnia, stomatitis, suppuration, scaling, swelling, irritability, medicinal taste, photosensitivity, lacrimation, leukocytosis, thrombocytopenia, toxic granulation and eosinophilia.
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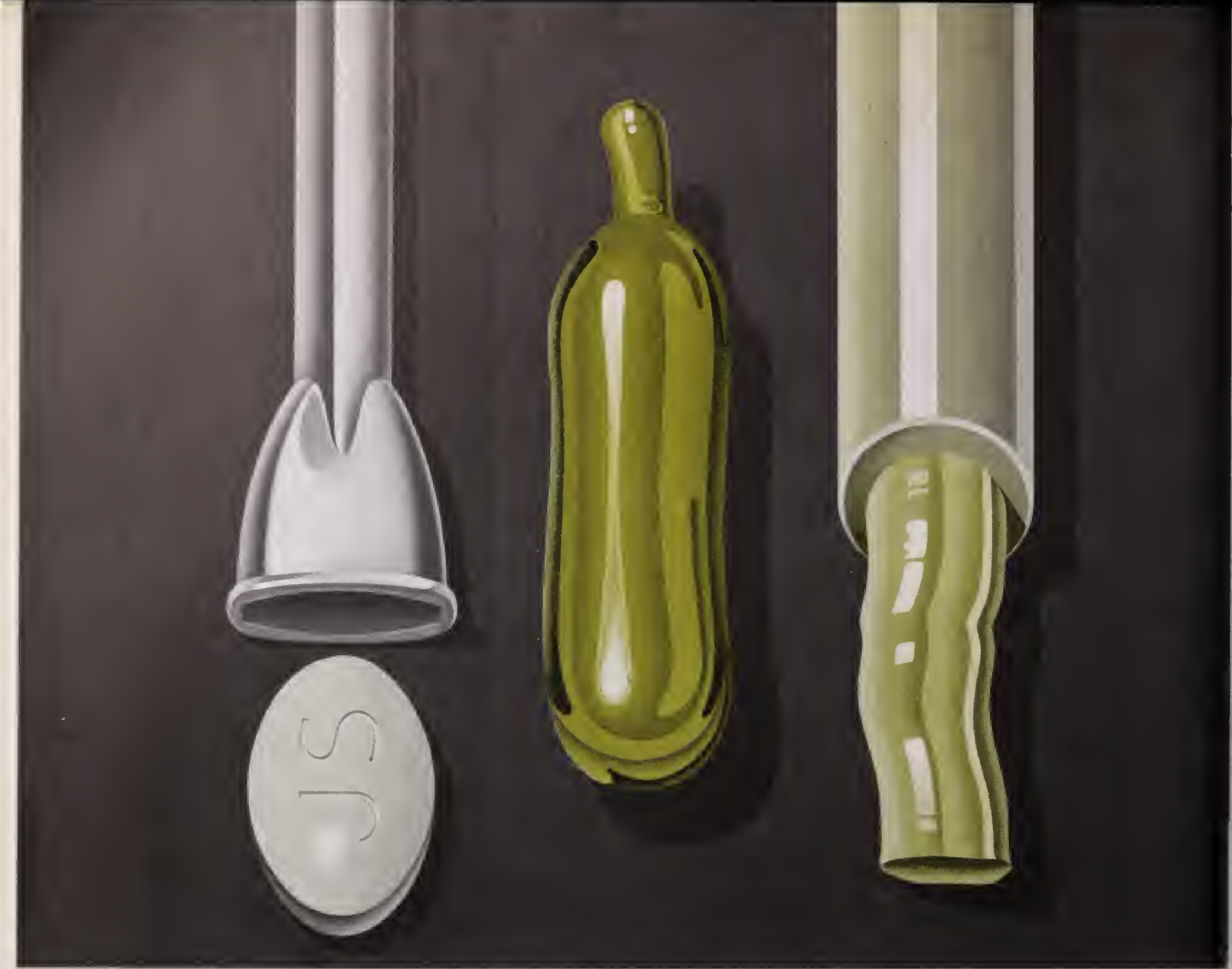

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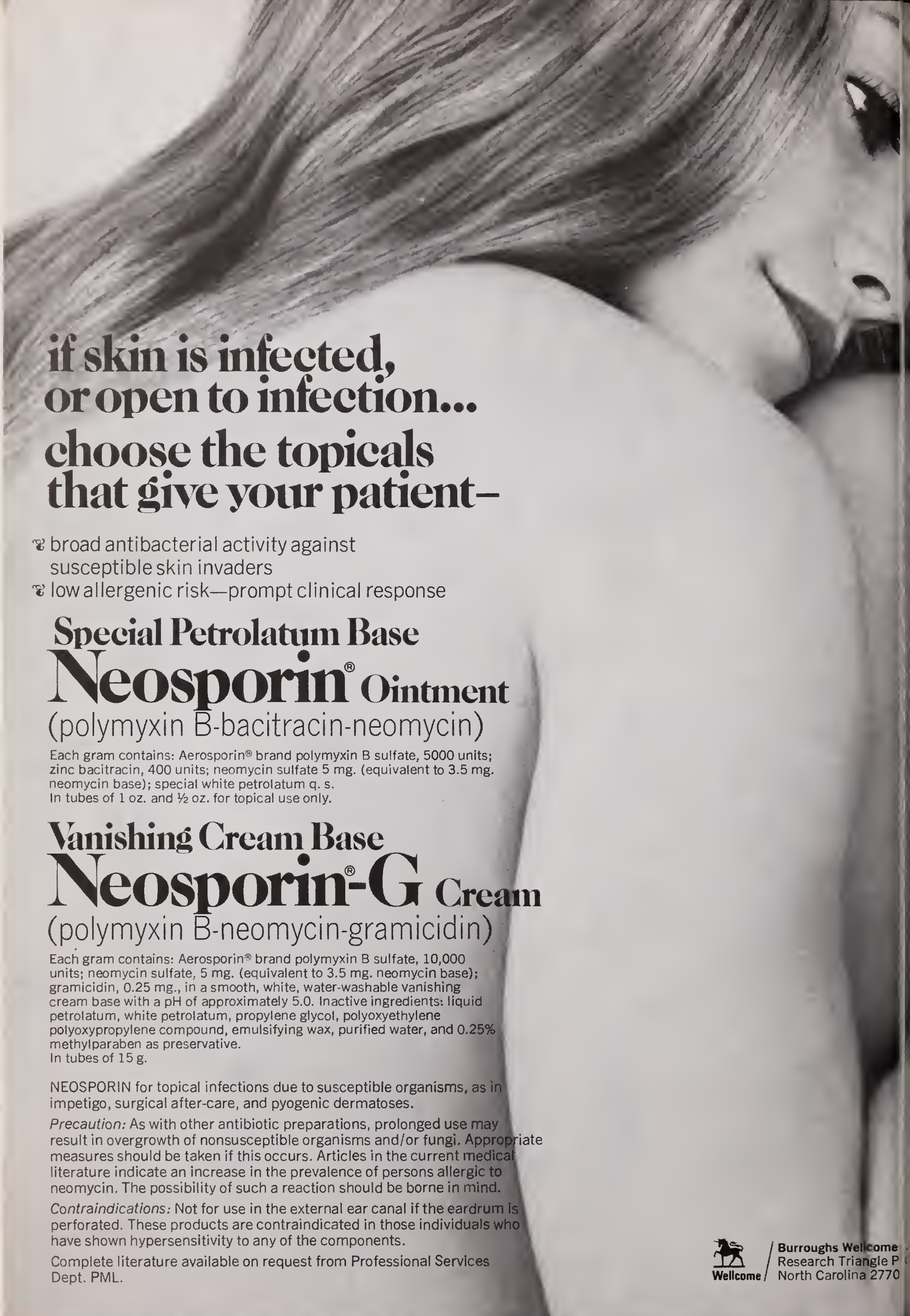

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PRESIDENT'S MESSAGE

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5 OCT 1972

CERTIFICATE OF NEED

"We seem to be in a stage of "entrapment by cliche, entrapment by stereotype."'

Harry Schwartz
The New York Times

rrell C. Crain, M.D.

Health planners who have wearied of upstaging ordinary mortals with such terms as "health care crisis", "HMO", "vendors of services", "marketing techniques", have a new shibboleth: "Certificate of Need". And that which a few years ago was only a little cloud like a man's hand over the sea of health facilities threatens to become a great rain washing away all individual physician's choice in the matter of medical practice.

Basically a Certificate of Need is a document indicating that an appropriate agency has investigated the need for a new health facility or for major changes in an existing one, and certifies that need for such a facility exists. Failing to obtain such a certificate, a proposed new facility could not be constructed nor could major changes be made in one already in existence.

Once confronted with such a proposition, the reaction of almost everyone is at first enthusiastically affirmative. The cost of constructing hospital beds and the frequent operating deficits which must be amortized by public subscription, or tax dollars, would seem to demand careful planning in regard to the location, size and type of new hospital constructions. But the problem is not so simple. If a hospital is to be constructed entirely with private funds, and any operating deficit will be met without public solicitation, must a Certificate of Need still be obtained? And in a community such as ours, what area is to be included? - the geographic limits of the District or the Metropolitan area? - and if this latter, how do we get the adjacent counties to cooperate? The District of Columbia Health Planning Advisory Committee, organized under the Department of Human Resources in our city government, exercises definate control over such matters in our city. Although its legal restraint when public funds are not utilized seems rather tenuous, and it has no authority beyond the city limits, the various agencies set-up to date

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These "paravenereal" diseases all have one thing in common: while they may be transmitted during the sexual act, their transmission is not always venereal. Thus, warts in the genital area may be spread from warts on nongenital skin. Candidiasis of the skin may result, not from sexual contact, but from contamination originating in the patient's own intestinal tract. In pediculosis pubis we have a disease that patients have been talking about for hundreds of years: a disease one can get from a toilet seat. The pubic louse is known to be able to survive for several hours without human contact and can therefore change hosts through an intermediate stop in a public restroom.

These diseases have a second trait in common: while they are under no obligation to be transmitted by sexual contact, this is their most obvious and common means of transmission. The physician is therefore obligated to perform three services. First, he must refrain from accusing his patients of sexual promiscuity. It is perfectly possible for a husband and wife to be completely faithful to each other and still acquire one of these "paravenereal" conditions. In this connection, I object to the use of the word "venereal" in talking about these diseases, since the connotation to the patient is one of obligatory venereal disease and sexual promiscuity. While this term may be applicable in some cases it is certainly not in others.

The second action one must take is to realize that these diseases are contagious during sexual relations. If treatment is to succeed, the patient's sexual partner or partners must be found and treated. Since contact history and case finding for these diseases are not done by the public health service, it is the physician's obligation to do so.

Lastly, one must consider the true venereal diseases. Since patients who are seen with paravenereal conditions of the genitalia are, for the most part, young and sexually active, they are potential candidates to develop true venereal diseases. For this reason, examinations may be indicated to rule out syphilis, gonorrhea, chancroid, and lymphogranuloma venereum in these patients.

Mervyn L. Elgart, M.D.

Associate Professor of Dermatology and
Associate Professor of Child Health & Development
George Washington University School of Medicine

References upon request

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Warnings: Use during pregnancy is to be avoided.

Precautions: 1. *Starvation Ketosis:* This must be differentiated from "insulin lack" ketosis and is characterized by ketonuria which, in spite of relatively normal blood and urine sugar, may result from excessive phenformin therapy, excessive insulin reduction, or insufficient carbohydrate intake. Adjust insulin dosage, lower phenformin dosage, or supply carbohydrates to alleviate this state. **Do not give insulin without first checking blood and urine sugar.**

2. *Lactic Acidosis:* This drug is not recommended in the presence of azotemia or in any clinical situation that predisposes to sustained hypotension that could lead to lactic acidosis. To differentiate lactic acidosis from ketoacidosis, periodic determinations of ketones in the blood and urine should be made in diabetics previously stabilized on phenformin, or phenformin and insulin, who have become unstable. If electrolyte imbalance is suspected, periodic determinations should also be made of electrolytes, pH, and the lactate-pyruvate ratio. The drug should be withdrawn and insulin, when required, and other corrective measures instituted immediately upon the appearance of any metabolic acidosis.

3. *Hypoglycemia:* Although hypoglycemic reactions are rare when phenformin is used alone, every precaution should be observed during the dosage adjustment period particularly when insulin or a sulfonylurea has been given in combination with phenformin.

Adverse Reactions: Principally gastrointestinal; unpleasant metallic taste, continuing to anorexia, nausea and, less frequently, vomiting and diarrhea. Reduce dosage at first sign of these symptoms. In case of vomiting, the drug should be immediately withdrawn. Although rare, urticaria has been reported, as have gastrointestinal symptoms such as anorexia, nausea and vomiting following excessive alcohol intake.
(B) 98-146-103-D (6/72)

For complete details, including dosage, please see full prescribing information.

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of moderate to severe intensity

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- **Not subject to narcotic controls:** convenient to prescribe – day or night – even by phone.
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50 mg. Tablets

Talwin®

brand of
pentazocine (as hydrochloride)

the long-range analgesic

a new outlook in chronic pain

of moderate to severe intensity

Contraindications: Talwin, brand of pentazocine (as hydrochloride), should not be administered to patients who are hypersensitive to it.

Warnings: *Head Injury and Increased Intracranial Pressure.* The respiratory depressant effects of Talwin and its potential for elevating cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions, or a preexisting increase in intracranial pressure. Furthermore, Talwin can produce effects which may obscure the clinical course of patients with head injuries. In such patients, Talwin must be used with extreme caution and only if its use is deemed essential.

Usage in Pregnancy. Safe use of Talwin during pregnancy (other than labor) has not been established. Animal reproduction studies have not demonstrated teratogenic or embryotoxic effects. However, Talwin should be administered to pregnant patients (other than labor) only when, in the judgment of the physician, the potential benefits outweigh the possible hazards. Patients receiving Talwin during labor have experienced no adverse effects other than those that occur with commonly used analgesics. Talwin should be used with caution in women delivering premature infants.

Drug Dependence. There have been instances of psychological and physical dependence on parenteral Talwin in patients with a history of drug abuse and, rarely, in patients without such a history. Abrupt discontinuance following the extended use of parenteral Talwin has resulted in withdrawal symptoms. There have been a few reports of dependence and of withdrawal symptoms with orally administered Talwin. Patients with a history of drug dependence should be under close supervision while receiving Talwin orally.

In prescribing Talwin for chronic use, the physician should take precautions to avoid increases in dose by the patient and to prevent the use of the drug in anticipation of pain rather than for the relief of pain.

Acute CNS Manifestations. Patients receiving therapeutic doses of Talwin have experienced, in rare instances, hallucinations (usually visual), disorientation, and confusion which have cleared spontaneously within a period of hours. The mechanism of this reaction is not known. Such patients should be very closely observed and vital signs checked. If the drug is reinstituted it should be done with caution since the acute CNS manifestations may recur.

Usage in Children. Because clinical experience in children under 12 years of age is limited, administration of Talwin in this age group is not recommended.

Ambulatory Patients. Since sedation, dizziness, and occasional euphoria have been noted, ambulatory patients should be warned not to operate machinery, drive cars, or unnecessarily expose themselves to hazards.

Precautions: *Certain Respiratory Conditions.* Although respiratory depression has rarely been reported after oral administration of Talwin, the drug should be administered with caution to patients with respiratory depression from any cause, severe bronchial asthma and other obstructive respiratory conditions, or cyanosis.

Impaired Renal or Hepatic Function. Decreased metabolism of the drug by the liver in extensive liver disease may predispose to accentuation of side effects. Although laboratory tests have not indicated that Talwin causes or increases renal or hepatic impairment, the drug should be administered with caution to patients with such impairment.

Myocardial Infarction. As with all drugs, Talwin should be used with caution in patients with myocardial infarction who have nausea or vomiting.

Biliary Surgery. Until further experience is gained with the effects of Talwin on the sphincter of Oddi, the drug should be used wit caution in patients about to undergo surgery of the biliary trac

Patients Receiving Narcotics. Talwin is a mild narcotic antagonis Some patients previously receiving narcotics have experienced mil withdrawal symptoms after receiving Talwin.

CNS Effect. Caution should be used when Talwin is administere to patients prone to seizures; seizures have occurred in a few suc patients in association with the use of Talwin although no cause an effect relationship has been established.

Adverse Reactions: Reactions reported after oral administratio of Talwin include *gastrointestinal:* nausea, vomiting; infrequentl constipation; and rarely abdominal distress, anorexia, diarrhe *CNS effects:* dizziness, lightheadedness, sedation, euphoria, hea ache; infrequently weakness, disturbed dreams, insomnia, syncop visual blurring and focusing difficulty, hallucinations (see *Acu CNS Manifestations* under WARNINGS); and rarely tremor, irr tability, excitement, tinnitus. *Autonomic:* sweating; infrequentl flushing; and rarely chills. *Allergic:* infrequently rash; and rarel urticaria, edema of the face. *Cardiovascular:* infrequently decrea in blood pressure, tachycardia. *Other:* rarely respiratory depressio urinary retention.

Dosage and Administration: *Adults.* The usual initial adult dose 1 tablet (50 mg.) every three or four hours. This may be increase to 2 tablets (100 mg.) when needed. Total daily dosage should n exceed 600 mg.

When antiinflammatory or antipyretic effects are desired in add tion to analgesia, aspirin can be administered concomitantly wit Talwin.

Children Under 12 Years of Age. Since clinical experience in chi dren under 12 years of age is limited, administration of Talwin this age group is not recommended.

Duration of Therapy. Patients with chronic pain who have receiv Talwin orally for prolonged periods have not experienced wit drawal symptoms even when administration was abruptly disco tinued (see WARNINGS). No tolerance to the analgesic effect h been observed. Laboratory tests of blood and urine and of liver a kidney function have revealed no significant abnormalities aft prolonged administration of Talwin.

Overdosage: *Manifestations.* Clinical experience with Talwin ove dosage has been insufficient to define the signs of this condition.

Treatment. Oxygen, intravenous fluids, vasopressors, and oth supportive measures should be employed as indicated. Assisted controlled ventilation should also be considered. Although nalc phine and levallorphan are *not* effective antidotes for respirato depression due to overdosage or unusual sensitivity to Talwin, pa enteral naloxone (Narcan®, available through Endo Laboratories) a specific and effective antagonist. If naloxone is not available, pa enteral administration of the analeptic, methylphenidate (Ritalin® may be of value if respiratory depression occurs.

Talwin is not subject to narcotic controls.

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pentazocine (as hydrochloride)

the long-range analgesic

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>
> *—George Sarton, from "The History of Medicine Versus the History of Art"*

Would it be useful in clinical practice to have government predetermine drugs of choice?

Opinion

Results of a survey of physicians:

13.3%
Yes, it would be useful.

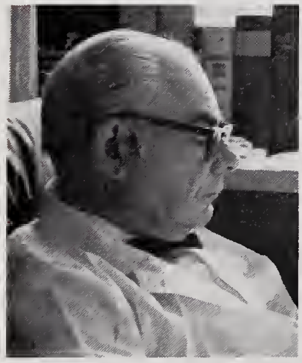
86.7%
No, it would not be useful.

Dialogue

Would it be useful in clinical practic to have government predetermine drugs of choice?

Doctor of Medicine

Walter Modell, M.D.,
Professor of Pharmacology,
Cornell University
Medical College,
Editor,
Clinical Pharmacology
& Therapeutics,
Drugs of Choice,
Rational Drug Therapy



The proposition that government should determine one or two "drugs of choice" within a given therapeutic class reflects the belief that a similarity in molecular structure insures a close similarity in pharmacologic effect. But this is by no means the rule. An obvious example would be in the field of diuretics, where a small change in chemical structure accounts for substantial differences in concomitant effects such as potassium excretion.

Any attempt to dictate the "drug of choice" would be complicated by the fact that some populations demonstrate a bimodal distribution in their reaction to drugs. If the data on drug response are mixed for the total population, one drug will appear to be as useful as the other. But if drug response is reported separately for different segments of the population, drug A will be found to be better for one group and drug B for the other.

It may, of course, be possible to determine drugs of choice in particular categories on a broad statistical basis. But there are always certain patients in whom a drug produces odd, unpredictable or idiosyncratic reactions. So, though a drug might statistically be the most useful one in a given situation, individual variations in response might make it the *incorrect* one.

The point I wish to make is that if two, three, four or more drugs in one class are of approximately equal merit, that in itself is justification for their availability. Exceptional cases do arise in which one drug would be useful to a certain segment of the population and another drug would be of no use at all. In the practice of medicine, the physician must be prepared to treat the routine as well as the unusual case.

Another objection to the determination of a drug of choice is that precise statements of *relative* efficacy are very difficult to make—much more difficult than statements of efficacy. For example, in testing drug efficacy, it is easy to determine the difference between a drug that is effective in treating a condition and one that is not at all effective. Thus, it is fairly easy to determine whether a drug is more effective than a placebo. But if you compare one drug that is effective with another drug that is also effective, and the relative differences between them are very slight, statements of relative efficacy may be very difficult to make with assurance.

I do not mean to imply that relative efficacy statements are not useful or can never be made. With some groups of drugs (e.g., analgesics), extensive study and precise methodology have yielded useful information on relative efficacy. But in most situations, such information can be acquired only through studies encompassing three to five years of use in many more patients than are used to compare drugs with a placebo for the introduction of a drug into commerce. It is really only after practitioners use a drug extensively that relative safety and efficacy in practice can reall
determined.

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Maker of Medicine

neth G. Kohlstaedt, M.D.,
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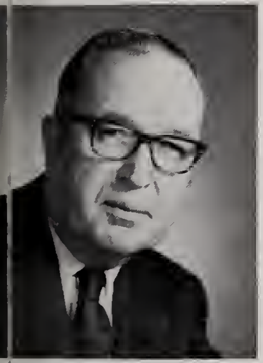

n my opinion, it is not
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that the established products should not be so easily dismissed.

Variation in patient response to drugs constitutes one of the major obstacles to the determination of "drugs of choice." We are just beginning to open the door on pharmacogenetics, but it is evident that genetic differences cause wide variations in the way drugs are absorbed, metabolized, etc. This fact alone is sufficient to make unrealistic the idea that there is one drug in each class to be used for every human being.

The problem of determining relative drug efficacy is an extremely complicated one. Comparison with other drugs of the same class should not be a prerequisite for marketing a new substance. In some therapeutic areas, it may be difficult to make accurate comparisons. For example, in the treatment of infections it is not possible to conduct crossover studies. Recovery may be influenced by factors which cannot be controlled or measured, i.e., natural host resistance and virulence of infective agents. A drug's acceptability must often be judged on the basis of its own performance, and this may be limited to experience in a relatively small patient population. If the introduction of a new drug must await the adequate establishment of relative efficacy, the duration of clinical trial and extent of studies would be greatly prolonged, particularly for rare or unusual conditions. The availability of a new drug would be delayed. Many patients might suffer needlessly and lives might be lost.

Relative efficacy can best be established by experience in a general patient population through regular channels of clinical practice. The physician considers the patient as a whole, which means the patient often has multiple problems and drugs must be selected with this in mind. Hence, a "drug of choice" in an uncomplicated case may not be the best drug for a patient with associated problems. Publication of well-controlled studies in medical journals may provide comparative evidence; discussions at medical meetings, presentations at postgraduate courses, and the new audiovisual technology may bring evidence to physicians on comparative therapy. In a free medical marketplace, a drug that does not measure up will fall into disuse. For example, broad clinical experience has established vitamin B_{12} as the "drug of choice" for the treatment of primary pernicious anemia. No amount of advertising or promotional effort by the manufacturer could increase the use of liver extract for this anemia. However, a physician may wish to employ parenteral liver preparations for a special purpose.

In the field of surgery, peer review in the hospital has brought significant improvement in the use of new techniques and procedures. Something of this nature would be useful in the area of drug therapy. However, it should be developed by the medical profession itself and would necessitate, for its proper function, an improvement in the dissemination of reliable data on clinical pharmacology of drugs under consideration.

Ideally, information on the relative efficacy of drugs should be gathered and assessed by the physicians who actually administer the specific agents to a specific patient population. To do this, they will need even more information on the drugs they use – information that the pharmaceutical manufacturers must begin to provide if government regulation of *"drugs of choice"* is to be avoided.

Opinion & Dialogue

**What is your opinion, doctor?
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Genetic Susceptibility to Deleterious Effects of Environmental Factors

VERLE E. HEADINGS, M.D., PH.D.*

An increasing concern in medicine involves the quality of man's environment as it relates to his health. For many years this concern dealt basically with those microbes which are deleterious to man or with relatively select situations, as for example, scrotal cancer among chimney sweeps. In recent times, however, certain risks related to environmental quality are virtually universal in industrialized societies because of the wide dispersion of increasing numbers of industrial products. The monitoring of all such products for potential long-term effects on health is a formidable task both because of the number of such products and the difficulties encountered in reliably quantifying given effects.

Given the large degree of genetic variation which occurs in an unselected population, it is to be expected that a significant number of individuals might be susceptible to some environmental factors. It is now recognized that a number of both rare and relatively common clinical disorders are either initiated or exacerbated by genetic susceptibility to certain agents in the affected individual's environment. Prevention of such disorders can be achieved either by identifying susceptible individuals through screening programs, followed by protection against the offending agent or by reducing exposure of the entire population to such an agent.

Genetic Variation

One of the more significant recent developments in genetics is the recognition that at least one-third of gene loci in human populations are polymorphic.[1] This means that at a given gene locus in a particular population there may be two or more alternative alleles, each with a frequency of 0.01 or greater. Each allele of a given locus produces a structurally distinct product which may possess functional properties differing from that of other allele products. In the mouse and in the fruit fly, both widely used species in genetic research, at least one-third of gene loci tested are also polymorphic.

A fundamental question and one which generates much debate concerns the mechanisms by which a genetic variant can achieve relatively high frequencies in a population. It is generally believed that the functional properties of a given variant either singly or in concert with products of other genetic loci confer a reproductive advantage on the affected individual. With current techniques it is difficult to demonstrate a structure–function–natural selection relationship for polymorphic human genes. A major exception is the hemoglobin S in which a specific amino acid substitution in hemoglobin makes the red blood cell subject to lysis when infected by *Plasmodium falciparum*. Lysis of such infected cells prevents intracellular maturation of the larva and thereby prevents malaria. In those areas of the world where malaria was endemic for many generations and a major cause of death among children, the protective effect of the sickle-cell trait resulted in an increase in the frequency of the gene for hemoglobin S.

Recent information on 4 enzyme systems in unselected populations of Drosophila indicates that those enzyme systems with high equilibrium constants contain larger numbers of genetic variants in the polymorphic frequency range.[2] This suggests that natural selection may operate more efficiently on the variants of those enzyme systems which catalyze relatively irreversible reactions.

Despite limited data on functional and selective advantages of polymorphic genetic variants, it is evident that some of these variants confer on individuals differing sensitivities toward environmental agents. This has been particularly well documented for responses to certain pharmaceuticals. Variability in response to drugs reflects at least three types of genetic variability:

1. Many drugs are processed in the body by a series of enzyme-mediated reactions, and each enzyme is potentially subject to minor variations in specific activity resulting from genetic

---

* Geneticist and Assistant Professor of Pediatrics, Howard University College of Medicine, Washington, D.C.

Preparation of this paper was supported by special project Grant 12HS: 414, U.S. Children's Bureau, to the Pediatrics Department of Howard University.

Reprint requests may be addressed to Dr. Headings at the Department of Pediatrics, Howard University College of Medicine, 520 W Street, N.W., Washington, D.C. 20001.

mutations of minor effect. Assuming that at least one-third of gene loci are polymorphic, a large variety of genetic combinations involving biotransformation of drugs are available in a given population. As a result of this variety a given quantitative property, such as half-life of the drug in blood or potency of the drug, occurs as a range of values in a population.

2. A genetic variant involving a single biotransformation enzyme may produce a major functional abnormality when the individual is exposed to a specific environmental factor. Representative of this category is an inherited deficiency in pseudocholinesterase. This deficiency is not deleterious to affected individuals unless the muscle relaxant, suxamethonium, is administered to them, whereupon they experience prolonged apnea.

3. A protein system which is normally insensitive to a particular class of drugs may be rendered susceptible by a genetic mutation which alters the structure of that protein. Illustrative of such susceptibility are those hereditary hemoglobin variants, which on exposure to sulfa drugs lead to methemoglobinemia and hemolysis, and glucose-6-phosphate dehydrogenase deficiency which produces episodes of anemia upon exposure to a variety of chemicals.[3]

Natural selection theory holds that those genetic variants which are favored by a given set of environmental conditions will achieve relatively higher frequencies in a population. Those variants which are incompatible with the environment will be eliminated or restricted to a low frequency. Much of current medical care is directed at regulating the private environment of individuals with genetic disorders so as to reduce their biological incompatibility with their environment. It is important to recall that throughout much of man's history environmental change has been a slow process, causing proportionately few individuals to suffer in any given generation from genetic maladjustment to environmental conditions. It is to be expected that a rapidly changing environment will subject increased numbers of individuals to environmental conditions for which they are genetically unprepared. The accelerating rate of change currently experienced by industrialized societies is unprecedented and therefore requires close scrutiny as to its potential interaction with the genetic composition of individuals.

Heritable Susceptibility to Environmental Factors

A number of diseases have now been described for which it is reasonably certain that individuals possess a major genetic susceptibility to selected environmental agents, frequently the products or by-products of man's industry (table I). The examples to be discussed include diseases in which the onset of signs and symptoms occurs during middle age, childhood, and in the fetus. Research with animal models has identified biochemical mechanisms for susceptibility to given environmental agents.

Alpha$_1$ –antitrypsin is an alpha globulin found in human serum which exhibits at least 10 genetically different variants.[4] One of these in homozygous form is characterized by about a 90% quantitative deficiency and is frequently associated with the onset of emphysema by middle age.[5] Evidence has also been presented for an increased risk of emphysema in the individual who is heterozygous for the deficiency.[5] Homozygous- and heterozygous-deficient individuals are estimated to occur in a frequency of about 0.7% and 5% respectively in certain Caucasian populations. It is commonly appreciated that certain air-borne pollutants, including that from cigarette smoking, increase the risk and severity of emphysema. Given these relatively high frequencies it is apparent that the widespread occurrence of certain industrial pollutants and the habit of smoking creates conditions to which significant numbers of individuals are genetically maladjusted.

Although not clearly defined in genetic terms or in gene-environment interaction terms, it is probable that occurrence of atherosclerosis requires genetic susceptibility and that the severity of the disease is likely exacerbated by certain dietary and exercise habits.

A form of inherited glucose-6-phosphate dehydrogenase deficiency confers susceptibility to acute hemolysis when the individual is exposed to any one of an array of widely used chemical substances.[3] These substances include acetanilid, sulfonamides, nitrofurantoin and related

TABLE I: Examples of Genetic Susceptibility to Environmental Factors

Disorder	Environmental Factor(s)
Drug-susceptible hemoglobins	Sulfa drugs
Glucose-6-phosphate dehydrogenase deficiency	Acetanilid Sulfonamides Nitrofurantoin Naphthalene (moth balls) Antimalarials Fava beans
Pseudocholinesterase deficiency	Suxamethonium
Emphysema (Alpha$_1$ -antitrypsin deficiency)	Cigarette smoking
Porphyria (barbiturate sensitivity)	Barbiturates
Lactose intolerance (lactase deficiency)	Lactose
Maternal rubella deafness	Rubella virus

antibacterial agents, naphthalene (moth balls), and antimalarials. The frequency of this x-linked enzyme deficiency is between 10 and 15% among Afro-American males. With regard to this one enzyme system, therefore, a significant fraction (about 0.5%) of the population of the United States is susceptible to deleterious effects of certain widely used products of the chemical and pharmaceutical industries. Rare deficiency variants of other enzymes in the hexose monophosphate shunt pathway of erythrocytes have also been reported to confer susceptibility to drug induced hemolysis.[6]

Since the description of thalidomide embryopathy in 1961[7] much of the information on teratogenic agents has dealt with interspecies and intraspecies differences in susceptibility. It has been estimated that approximately only 20% of women who ingested thalidomide in teratogenic doses during the sensitive period of the 34th to 50th days of gestation gave birth to malformed infants.[8] Dosage sensitivity for thalidomide teratogenicity in rhesus monkeys and baboons is similar to man[9] and for rats and hamsters is estimated to be 100- and 700-fold less, respectively.

A chlorinated hydrocarbon, the defoliant 2, 4, 5-T, is teratogenic in rats, mice, hamsters and chickens when applied in relatively large doses.[10,11] Although relatively low teratogenicity is claimed for this compound in these species, the occurrence of differences between species in susceptibility to teratogens in general is cause for concern in human populations. Indeed, preliminary information on types and incidence of congenital malformations in human populations of S.E. Asia which were exposed to this agent indicates that this and similar chemicals deserve closer scrutiny. Differences in genetic susceptibility to this teratogen among individuals within a species must also be considered, since this has been demonstrated for other teratogens.

In mice administration of certain steroids during pregnancy induces cleft palate in the fetus. Of particular interest to this discussion is the observation that susceptibility to this teratogenic effect differs markedly among various inbred strains.[12] Among outbred populations genetic differences in susceptibility to an agent are frequently difficult to detect unless careful family studies are done. This was the case for one component of rubella embryopathy, deafness.[13] Among the pedigrees of children in whom a diagnosis of maternal rubella deafness was made there occurred an increased frequency of deafness and in deficiency of both hearing threshold and stapedius reflex.

Application to Health Care

Given our present state of technology, diseases with a significant environmental component are technically more amenable to prevention than are those wherein the genetic determinant is the major element. The strategy for preventing diseases of genetic-environment origin includes both identification of genetic susceptibility and the environmental agent which either initiates or exacerbates symptoms.

There are a minimum of four approaches to this area of health care:

1. A key responsibility rests with the practicing physician at the point of a high level of suspicion for disorders of gene-environment interaction, followed by obtaining detailed family history and history of environmental contacts. Historically, associations between a clinical disorder and its etiology have been initially described in this way, as exemplified by the identification of X-rays, rubella, thalidomide and mercury as being teratogenic.

2. Screening for genetic susceptibility to specific disorders with a significant environmental component is becoming a possibility. A notable example is the identification of individuals with serum alpha$_1$-antitrypsin deficiency prior to the onset of emphysema. This permits the physician to offer both measures which can control exacerbating elements of the individuals environment, as well as genetic counsel, at a stage when such measures are most useful. We can anticipate that an increasing number of genetic disorders which can be ameliorated by environmental controls will be subjected to large-scale screening.

3. It is envisioned that in some instances the protection of the relatively few susceptible individuals can be assured only by controlling the exposure of an entire population to a given agent.

4. Testing industrial products for chronic effects on health, including teratogenicity and carcinogenicity, is obviously a necessity. A significant number of chemical products have been found, after they were introduced into man's environment, to exert deleterious effects. This suggests that in some instances more rigorous standards of testing than are presently in effect are desirable.

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THE VIEW BOX

Prepared by the

Department of Radiology

Washington Hospital Center

This 30-year-old Negro male presented clinically with a three week history of painless hematuria. He denied any recent trauma, flank pain or past history of venereal disease. Physical examination was within normal limits; no abdominal mass was palpable. Urinalysis showed *4+* RBC's.

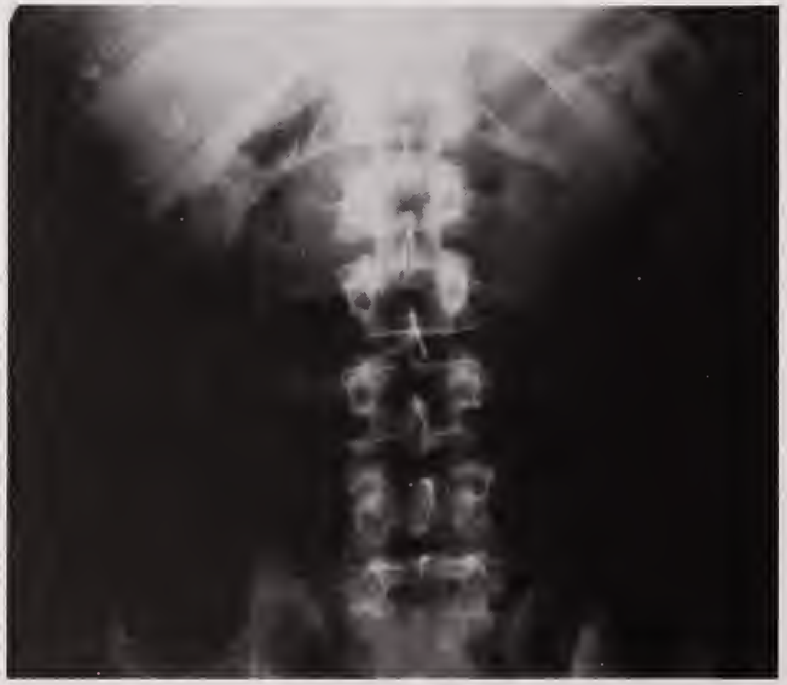

Fig. I Scout Film of Abdomen

(For diagnosis see page 564)

Organophosphate Insecticide Diazinon^{R}* Poisoning in Children

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Organophosphate compounds were originally prepared as pesticidals, but during World War II they were developed for biological warfare.[1] Later on these compounds were utilized as insecticidals, mainly in industry and agriculture. The less toxic forms of organophosphates are now finding use as pesticides for household purposes. One factor for their increased use is their quick hydrolysis and destruction. They lack any accumulative effects such as found with DDT (2,2,bis[p-chlorophenyl] 1,1,1-trichoroethane) and chlorinated hydrocarbons. They do not enter in the biological magnification cycle. Doctors and the public need to be alerted for an increase in incidence of accidental organophosphate poisoning in children due to improper storage and use of these agents in the home.

Organophosphates are anticholinesterases by their specific combination with cholinesterase to form a more stable phosphorylated cholinesterase. The acetylcholine thus remains unhydrolysed and so results in enhanced transmission of all preganglionic autonomic fibres, post-ganglionic parasympathetic nerve fibres, and all somatic motor nerves and some synapses in central nervous system. Insects have sudden death due to respiratory muscle contraction and later flaccid paralysis. In humans, acute and chronic poisoning has been reported in detail by Namba,[2] Hayes,[3] and more recently by Rapkin et al.[4]

The main effects are drug- and dosage-dependent and include, in the eyes, mieosis and conjunctival congestion; in the respiratory system, bronchial constriction and increased bronchial secretions; in the cardiovascular system, bradycardia, vasodilatation and resultant hypotension, coupled with sympathetic stimulation and sometimes an increase in blood pressure; in the gastrointestinal tract, vomiting, cramps, diarrhea; and in the central nervous system, disorientation, slurred speech, coma, dizziness, convulsion and central respiratory depression, and sweating.

The organophosphate insecticides include tetraethyl pyrophosphate (TEPP), Parathion, Systox, Phosdrin, Methyl Parathion, Octamethylpyrophosphate (OMPA), Dicyston, Guthion, O-Ethyl O-p-nitrophenylphosphonothicate (EPN), Trithion, Diazinon, Chlorothion, and Malathion. These compounds are highly toxic. With TEPP rated on a scale as 1,000, Diazinon would be rated as relatively less toxic at 2. The toxic effects of these compounds can result from inhalation, absorption from gastrointestinal tract, or absorption through the skin. The toxic effects are further complicated because organophosphates are turned into a more lethal form by the liver.

We are reporting a case of Diazinon® ingestion which was treated at D. C. General Hospital. Case report #391399. A 5-year-old Negro girl was brought to the outpatient department with complaint of dizziness and generalized weakness. She had been, apparently, in good health until 1-½ hours before admission, when she ingested an unknown amount of liquid from a "7-Up" bottle. The child vomited after 5 minutes and felt weak and dizzy. The mother volunteered the information that she got the insecticide solution from the landlord, which was kept in the "7-Up" bottle.

The patient's birth, past and family history was unremarkable.

The child, upon physical examination, was found to be completely disoriented, flushed, and irritable. The pulse rate was 100/minute and regular, the respiratory rate 52/minute with moderate subcostal retractions, and the blood pressure 100/70. She had excessive salivation. The pupils were pinpoint but reacted to light. The lung fields were full of moist rales. She had slurred speech. There were generalized fasiculation of all of the musculature of extremities, predominantly in the lower limbs.

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* O, O-diethyl O-2-isopropyl-4-methyl-6-pyrimidinyl phosphorothioate

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Fortunately, the resident in the emergency room recognized the symptoms of organophosphate insecticide poisoning. A careful gastric lavage was done. Intravenous fluids, ½ normal saline, and parenteral atropine was administered. First dose was intramuscular, but later 1-1.5 mg atropine was given intravenously at approximately 10-minute intervals. Atropinization was achieved after a total of 15 mg of atropine. This atropine dose was repeated whenever the atropine effect, i.e., flushings of skin or a heart rate of 120/minute, subsided. The lungs remained clear and salivation stopped. The patient continued to have fasiculation of the skeletal muscles. They were finally brought under control 5 hours after admission by a total of 700 mg of Pralidoxime, given intravenously.

Twelve hours after admission the child required only 1 mg of atropine every 2-5 hours. Following therapy she became alert and oriented. Atropine was discontinued after 48 hours. The child was discharged and seen a week later in the clinic; she was found to be in good health. She was given a prophylactic course of antibiotics during the hospital stay.

Laboratory data: Hemoglobin AA; serology for syphillis–negative; hematocrit 38%, WBC 29,900 the first day and 14,400 on the second day; urine negative except for 3+ glucose, 8-10 RBC and 6-8 WBC per high field, with many bacteria; BUN 12 mg%; glucose 260 mg%; K 2.2 mEq/L, Cl 102 mEq/L, Na 133 mEq/L, CO2 22 mEq/L, SGOT 59 units; thymol turbidity 6.3 units, and alkaline phosphatase K.A. units.

Discussion

Organophosphate poisoning has been reported in worldwide literature,[2] mainly due to their wide agricultural and industrial usage. In the United States, California reports nearly 300 cases per year.[3] Poisoning in children is less common, but this may change in the near future with increased domestic use. Organophosphates are not innocuous compounds. Many deaths have been attributed to them. The cause of death is mainly due to respiratory arrest, which may be a combination or individually attributable to paralysis of the respiratory muscles, failure of the respiratory center, or intense broncho-constriction with increased bronchial secretions. Hence, mechanical ventilators should be in readiness, and in some cases tracheostomy has been life-saving.

The diagnosis of organophosphate poisoning can be determined by a reliable and accurate history of exposure and by clinical manifestations. These symptoms can be confirmed by atropine tolerance. However, laboratory diagnosis can be made by measurements of serum cholinesterases; a severely ill patient will have 0-10% of normal activity of cholinesterases, a moderately ill patient 11-20%, and a mildly ill patient 21-50% activity. This test, unfortunately, was not available to our patient and is available only in highly specialized centers.

A urinary metabolite of organophosphate compounds is para-nitrophenol. This compound can be determined in the urine easily, but it just implies exposure and gives no good correlationship with intoxiation.

The physiologic antidote is atropine. This controls the parasympathetic stimulation. The specific antidote is Pralidoxime, which releases phosphate from cholinesterases. Pralidoxime is given intravenously, 25-50 mg/Kg (adult dose 1 gm) by slow infusion. This dose may be repeated as a continuous infusion in cases of severe poisoning.

Our patient was exposed to one of the less toxic forms of organophosphate compounds, Diazinon. Fortunately, this was diagnosed early and with the institution of proper therapy she recovered without any ill-effects.

Summary

A non-fatal case of organophosphate poisoning due to Diazinon in a child has been presented. The patient was treated with atropine and Pralidoxime. The incidence of organophosphate poisoning in children is probably going to increase due to their wider use in homes. This, however, can be prevented by a greater public awareness. Parents should be cautioned regarding storage of toxic agents in proper containers and should follow the instructions on the label.

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Lead Poisoning: A Crisis of Conscience*

ROGER S. CHALLOP, M.D.†

> *"No over-all statistics of any value are available. Nothing short of a long and costly campaign of enlightenment can bring any substantial change in this situation, to which ignorance, apathy, misdiagnosis, and the fact that lead poisoning is not a reportable disease are all contributing factors."*

I believe that we all would agree that this statement about childhood lead poisoning sums up todays problem very well. Unfortunately, it also very nicely summed up the same problem at the time it was written in 1953 in a book on accident prevention. Today's seminar would have been unnecessary if action had matched rhetoric then.

Lead poisoning has indeed been a "silent epidemic," affecting a population with little economic or political recourse for redress. It is not a "new disease," nor is it really an epidemic. It has not really spread, or come in waves, as have ancient plagues or our recent venereal disease upsurge. It is indeed silent, sitting hidden in the old paint of walls, windowsills and woodwork.

The medical profession today faces many challenges, some deserved, but many which are really directed at it as an accessible symbol of "the establishment." It was in response to some of these feelings that Henry L. Freund, Jr., Staff Attorney of the Legal Aid Society of the City and County of St. Louis, was led to comment at a discussion of the political and economic perspectives of lead poisoning: "Lest I be misunderstood, I believe the legal profession and legal system surpasses the medical field in its insensitivity and unresponsiveness to the needs, problems, and life styles of the poor."

For too long lead poisoning has been a "medical," more specifically a "medical-student's disease," to be handed from big city house staff to big city house staff but rarely to be carried to the field, upon entering practice or moving to small-town hospitals. It is here that the medical profession must bear the most responsibility. For, while reducing the mortality rate below 5% with advances in chelation therapy, there was little attempt to involve the "doers," i.e., the public, the legal profession, the politicians, and the housing technologists.

The political process and the whole arena of publicity-seeking is foreign to and in some ways thought to be out of line with traditional professional conduct. This "conspiracy of silence" perpetuated the lack of awareness of lead poisoning as any more of a problem than in the old central cities of the northeast. The medical professions in these cities did much to detect and treat children with lead poisoning. Without public awareness, housing correction, or sufficient funds, they were doomed to the status quo.

The medical profession alone cannot "cure" this disease. It is basically a housing and political problem. Medical input is needed in designing and manning screening and treatment facilities, as well as monitoring the results of long-term subclinical lead poisoning.

I believe that the corner has been turned. Medical awareness of lead poisoning as well as of its role in the political process is at an all-time high. Doctors have been at the forefront of recent local lead poisoning activities throughout the country. Medical students and nurses are being stimulated into joining community-related projects. Courses in community medicine are being given in medical-school and residency-training curricula.

Within the past year the subject of childhood lead poisoning has received much publicity, and suddenly "a silent epidemic" has been discovered in our midst. The time is now right to broaden the fight, to extend the responsibility for the physical and mental well-being of 2½ million children below the age of six living in deteriorating housing to the other professions that can effect change. The time is right for lawyers and judges to enforce housing codes and protect those children who are too poor to protect their constituted rights; for politicians and office holders, to seek passage of new, more workable ordinances and appropriate measures; for sanitarians, housing technologists, unemployed NASA engineers to lend their skills; and, most important, for parents and community groups to become more involved in their own destiny. If this problem is to be solved, it is at the community level that the action will occur.

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* Presented at the Lead Poisoning Seminar of the Medical Society of the District of Columbia October 18, 1971.

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It is perhaps true that to the ghetto mother surrounded by rats, crime, hunger, and a lack of municipal services, lead poisoning seems very minor. As a physician, I submit that a disease whose etiology, population group, and treatment is known must be dealt with. Mental retardation and death, although reduced, still occur. One day after a local critic described recently discovered cases of elevated blood leads in Aurora, Illinois, as "sick tests, not sick children," one of those "sick tests" died.

The number of cities having "discovered" a lead poisoning problem grows daily. It is quite apparent that it is not geography, but age of housing that determines the potential for lead poisoning. As part of a small sample neighborhood survey of high-risk children (i.e., substandard pre-World War II housing) done in 17 cities around the United States, an average of 10% of those screened had levels above 40 mcgm% and 5% were above 50 mcgm%. If only those between the ages of 1 and 3 years were tallied, the results would double. In almost any survey of high-risk neighborhoods, 5-10% of those screened were above 40 mcgm%, and in some as high as 45% above 40 were found. Many cities use a level of 60 mcgm as a cut-off because of the potentially dangerous assumptions of lower risk and economic unfeasibility in handling those below 60. New York City had 20,000 children above 40 mcgm among the 80,000 screened in 1970.

The Surgeon General recommends following those children between 40-49 mcgm but saving the confirmatory testing and possible treatment for those above 49 mcgm. The whole problem of detection and diagnosis has received much recent research and attention, resulting in the development of more accurate blood-lead techniques.

While blood-lead determinations remain the most consistent indicator of body-lead burden, the problem of laboratory standardization persists. The experience of a survey of 60 laboratories' measurement of blood-lead samples reported in the American Industrial Hygiene Association Journal of July-August, 1970, reflects this. Only 40% of the laboratories that participated, using macro samples, reported results of acceptable precision. To minimize laboratory errors, standard blood samples prepared by an established reference laboratory should be used by laboratories participating in child blood-lead analysis. This method has worked well in early field trials of reference samples prepared for our Bureau by Dr. Julian Chisolm and sent to several laboratories doing blood-lead analysis. The determination of blood leads must be done in a laboratory that does these routinely, but is still only a biochemical result that must be combined with clinical judgment.

Several new methods of microblood-lead determinations, as well as field wall-lead detectors, have made mass screening of children and houses a possibility. Modifications of atomic absorption spectroscopy as well as a new anodic stripping voltammetry technique have made finger-puncture blood-lead determinations a reality. Early field-test results, especially with the anodic stripping technique, have given fairly good screening results, but they still require more experience. Confirmatory venipuncture blood-lead determinations should and are being done by traditional techniques on all positives found. The dividends to be paid in patient acceptance and in fewer physicians being needed for finger-prick sampling is obvious.

Wall-lead detectors, employing the X-ray fluorescence principle, are portable and non-destructive devices to analyze lead on flat, painted surfaces. In the above-mentioned survey of 17 cities, 65% of the dwellings in the survey had potentially hazardous concentrations of lead on at least one accessible interior surface. While lead on the walls does not necessarily mean that the child will get lead poisoning, any normal child in the oral exploratory stage, between 1 and 3 years of age, will put any accessible object in his mouth. Accessibility, not esthetic factors, determines what a child eats. Having leaded paint, especially when the mother is unaware of the danger, presents the child with an avoidable hazard. Most cases of lead poisoning, encephalopathy and brain damage occur in this age group. Although low socio-economic status accounts for most cases in this group, because they are living in old substandard housing, no child is immune. Many middle-class blue-eyed, blond-haired children living in older housing have nibbled lead paint, even from "tight surfaces." Urban migration patterns from central-core cities to the suburbs have correlated well with an increased incidence of lead poisoning in previously unreported areas, as older housing is subdivided into apartments.

Wall-lead analyses are useful in both legal and medical aspects. Because lead readings are given in mg per square cm, a more accurate determination of the actual total accessible lead content is made, rather than a mere percentage. The lead content per unit area should be considered in relation to the maximum daily permissible intake of lead without excessive accumulation of body lead burden in children.

Recent work by Dr. Barry King indicates the DPI to be 300 mcgm of elemental lead from all sources. Daily ingestion of 1 cm square of two layers of 1% lead paint would give a dose equivalent to the DPI. Recent clinical evidence of children indicating strong pica activity show they eat as much as 11 square cm of paint chips per 24 hours. All this evidence indicates that a child exposed to high lead-paint content can, in a matter of 3 months, reach elevated blood-lead levels. Furthermore, once these levels are reached, transition to symptomatic lead poisoning can be rapid and unpredictable. With 30 million housing units built prior to World War II still in use, the frightening potential exists.

Lead poisoning is still not a reportable disease in over 40 states, despite the fact that childhood lead poisoning has occurred in at least 35 states. Because of the advent of microblood techniques and wall-lead analyzers, the sheer magnitude of the problem of screening should not be insurmountable. Physician and community awareness is necessary for the success of any screening attempt, but the battle will be won only if the cycle is broken through housing correction. It is in this area that lawyers and local and federal officials must be recruited. To do this, physicians must no longer condone their "crisis of conscience."

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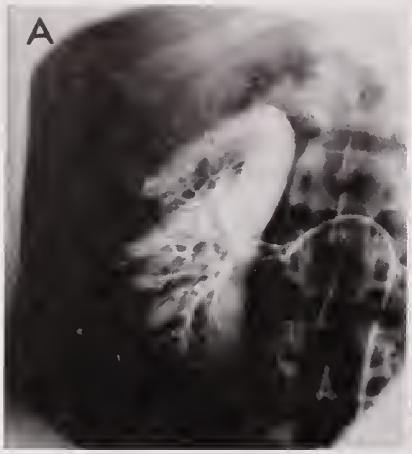
THE VIEW BOX

(from page 558)

DIAGNOSIS: Renal Cell Carcinoma (Hypernephroma, Grawitz Tumor)

The Drip Infusion Pyelogram showed a partially calcified 9 cm. mass lesion arising from the superior lateral aspect of the right kidney with minimal compression deformity of the superior calyceal group. A selective right renal arteriogram shows tumor vessels within a cystic mass. The renal vein appears normal.



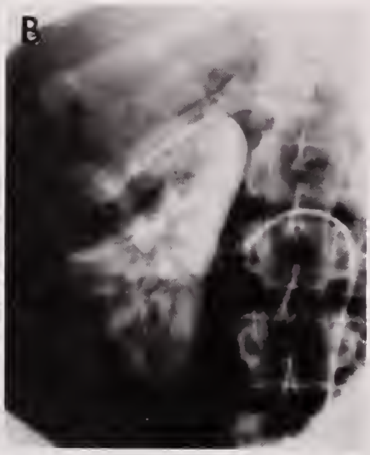


FIG 2a **FIG 2b**

At surgery this tumor was localized to the right kidney without invasive involvement of the adrenal gland or the liver. The pathology report was a necrotic renal cell carcinoma of the upper pole with diffuse calcification. There was no involvement of the renal vein.

Discussion: This tumor arises from epithelial cells of the renal tubules and accounts for 80-85% of all malignant renal tumors. Calcification is present in 30% of cases.

Age: most common above the age of 40.

Sex: twice as common in males as females

CLINICAL SYMPTOMS: often a triad of HEMATURIA (60%)
RENAL PAIN (50%)
RENAL MASS (20%)

May also have a fever, weight loss, sudden onset of hypertension, anemia or a varicocele that develops later in life.

Metastasis: usually the result of renal vein invasion.

1) local lymph nodes
2) lung and often to pleura (is the most common metastasis to pleura)
3) bone (osteolytic)
4) right heart, liver
5) brain, adrenals, and to opposite kidney

RADIOLOGIC FINDINGS: Mass lesion, deformed caliceal system on the IVP and retrograde. Thick walled sign in nephrotomograms in partially necrotic centers. Calcification where present in necrotic areas is usually linear or stippled in appearance. The most diagnostic procedure is ANGIO: lesion is often very vascular with A-V shunts and puddling of contrast material. Vena cavogram can be done to exclude or evaluate tumor thrombi prior to surgery.

A differential diagnosis would include inflammatory disease (abscess), renal cyst, hematoma, hematoma, and metastatic disease.

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Ventricular Tachyarrhythmias Controlled by Ventricular Aneurysmectomy and Aortocoronary-Saphenous Vein Bypass Grafts.

Report of a Case*†

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Despite the long-standing knowledge of the association of ventricular tachyarrhythmias and ventricular aneurysms with coronary artery disease, only nine cases could be found in the literature[1-6] describing control of ventricular tachyarrhythmias by ventricular aneurysmectomy in patients who have coronary artery disease. Aneurysmectomy is performed usually in patients who have congestive heart failure or thromboembolic episodes refractory to medical management. In order to further emphasize an additional indication for ventricular aneurysmectomy, we have described a patient with coronary artery disease and recent myocardial infarction who had recurrent ventricular tachyarrhythmias intractable to medical therapy and who became asymptomatic following ventricular aneurysmectomy and aortocoronary artery saphenous vein bypass grafts.

Report of Case

The patient was a 45-year-old Caucasian Navy man who first experienced chest pain in May 1971 while in transit across the United States. He was then hospitalized with the diagnosis of acute myocardial infarction. His recovery period was marred by several episodes of recurrent chest pain without documented enzyme rise, although there was electrocardiographic evidence of subendocardial ischemia. In August 1971 the patient had an episode of ventricular fibrillation for which resuscitation was required. During the next month he continued to have self-limited episodes of ventricular tachycardia which were not suppressed with intravenous lidocaine (4 mg per minute), procaineamide (500 mg every 3 hours, by mouth) and propranolol (40 mg every 6 hours, by mouth). In September 1971 the patient again had an episode of ventricular fibrillation, and resuscitation was required. At this point a temporary transvenous cardiac pacemaker was inserted to suppress the ventricular irritability. At that time there were neck-vein distension at 30°, bilateral basilar rales, an atrial gallop, and a grade II/VI holosystolic murmur at the apex which was transmitted to the left axilla. The ECG disclosed an old inferior myocardial infarction and diffuse repolarization abnormalities. Chest X-ray films showed a slight increase in vascular markings. The cardiac silhouette was slightly enlarged but was not diagnostic of ventricular aneurysm.

Retrograde cardiac catheterization was performed on September 30, 1971. The left ventricular end-diastolic pressure varied from 16 to 32 mm Hg depending upon the relationship of the paced QRS complex to the preceeding atrial contraction. The left ventriculogram showed mild mitral insufficiency and apical and septal areas of akinesis. Selective coronary arteriography disclosed complete occlusion of the right coronary artery and 90% occlusion of the left anterior descending coronary artery.

On October 5, 1971 the patient underwent cardiac surgery. During this procedure an akinetic portion, measuring 7 x 3.5 cm, of the left ventricular free wall was excised. In addition, saphenous-vein grafts were interposed between the aorta and right coronary artery and another between the aorta and anterior descending coronary artery.

The postoperative course was satisfactory. Three weeks following the

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[†] Read in part before the Washington Heart Association, Washington, D.C., on December 7, 1971.

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The opinions or assertions contained herein are those of the authors and are not to be construed as official or as reflecting the views of the Medical Department of the Navy or the naval service at large.

operation the cardiac rhythm was monitored for a 10-hour period immediately before discharge. The only arrhythmia demonstrated was occasional isolated premature ventricular contractions. Eight weeks postoperatively, however, while home on leave, the patient suffered recurrent ventricular tachyarrhythmia and was hospitalized. Resuscitative efforts were unsuccessful, and the patient died several hours after admission.

At necropsy the saphenous-vein graft to the right coronary artery was seen to be thrombosed, probably secondary to cicatricial contracture at the aorto-saphenous vein anastomosis. The vein graft to the left anterior descending coronary artery was patent. Thrombus was not present in the left ventricle.

Discussion

Since Lewis[7] produced ventricular tachycardia in dogs in 1909 by ligating the coronary arteries, this arrhythmia has been attributed frequently to coronary artery disease. Scott[8] successfully controlled ventricular tachycardia with quinidine in 1921. Since that time a variety of pharmacologic agents have been used with varying degrees of success to control ventricular tachyarrhythmias in patients with coronary artery disease. In 1964 artificial cardiac pacing was used successfully to control recurrent ventricular tachycardia.[9] Friedberg[10] reported the case of a 48-year-old man with a previous myocardial infarction and ventricular aneurysm who developed recurrent ventricular flutter and fibrillation which were unresponsive to various drugs. Transvenous cardiac pacing at a rate of 150 beats per minute, combined with antiarrhythmic drugs, was partially successful.

The importance of abating ventricular tachyarrhythmias in patients with coronary artery disease is well recognized. In a series compiled before our present mode of therapy became available, paroxysmal ventricular tachycardia in patients who had coronary artery disease had a 42% mortality rate over a period of six months. When the course of illness was further complicated by acute myocardial infarction the mortality rate increased to 64% over the 30-day period following onset of this arrhythmia.[11]

Ventricular aneurysm is reported to occur in from 10% to 40% of myocardial infarctions.[12] The first successful postmyocardial infarction ventricular aneurysmectomy without cardiopulmonary bypass was reported in 1955.[13] Indications for ventricular aneurysmectomy have included intractable congestive heart failure, systemic embolization refractory to anticoagulant therapy, and more recently, ventricular tachyarrhythmias.[6]

Several mechanisms by which ventricular aneurysms might promote recurrent ventricular tachyarrhythmias have been suggested. Rapid pulse formation originating from abnormal aneurysmal tissue may lead to ventricular tachyarrhythmias. Another suggested mechanism is based on myocardial ischemia. Myocardial oxygen consumption is increased in the presence of ventricular aneurysm because of increased left ventricular wall tension due to the enlarged left ventricle (Law of Laplace). In the face of compromised coronary arterial perfusion, as seen in the patient with coronary artery disease, a ventricular aneurysm can induce further ischemia leading to ventricular tachyarrhythmias.

Recently a case of formerly intractable ventricular tachycardia was controlled by coronary re-vascularization together with plication of an aneurysm on the diaphragmatic surface of the right ventricle.[14] This case resembles ours in many features.

Despite therapy with antiarrhythmic drugs and "overdrive" (higher) cardiac pacing we could not prevent recurrent ventricular tachyarrhythmias in our patient. It was only after resection of the aneurysmal portion of the left ventricle and direct re-vascularization of the ischemic left ventricle that the tachyarrhythmias abated. Suggested mechanisms by which ventricular aneurysmectomy may control ventricular tachyarrhythmias have been discussed. Besides resecting the aneurysmal portion of the left ventricle, coronary re-vascularization was accomplished. The approach to treatment of this patient was based on the hypothesis that removal of abnormal myocardial tissue, reduction of myocardial oxygen consumption, and improvement of coronary arterial perfusion may each contribute to the control of the ventricular tachyarrhythmias. Although prolonged survival is not claimed in this case, the prompt control of formerly intractable ventricular arrhythmias by the operation evidences the validity of these concepts.

Since only a limited number of cases have been reported in which ventricular aneurysmectomy successfully controlled ventricular tachyarrhythmias, our case is reported to further support this form of therapy. In addition to ventricular aneurysmectomy, direct myocardial re-vascularization should be considered for patients who have coronary artery disease and a ventricular aneurysm. Myocardial re-vascularization surgery in selected patients has the potential of controlling otherwise intractable ventricular tachyarrhythmias; also, long-term benefits of improved coronary blood flow in patients with ischemic heart disease may include increased longevity, diminished incidence of re-infarction, and relief of angina pectoria. This report supports the concept recently introduced into the literature[15] that patients requiring resection of a ventricular aneurysm should undergo selective coronary arteriography pre-operatively to determine whether myocardial re-vascularization surgery should also be accomplished.

Summary

A patient who had coronary artery disease and recent myocardial infarction manifested recurrent ventricular tachyarrhythmias unresponsive to medical management, including "overdrive" stimulation by a transvenous artificial cardiac pacemaker. It was only after he underwent ventricular aneurysmectomy and surgical myocardial re-vascularization that the ventricular tachyarrhythmias abated. Relatively few cases have been reported in which ventricular tachyarrhythmias have been controlled by ventricular aneurysmectomy. After approximately two months of effective therapy ventricular tachyarrhythmias recurred and the patient died.

This operation has been reserved traditionally for the patient who has intractable congestive heart failure or systemic embolization. Besides this case, there is 1 other in the literature in which surgical myocardial re-vascularization has contributed to the control of ventricular tachyarrhythmias. Possible mechanisms responsible for the production of ventricular tachyarrhythmia in patients who have coronary artery disease and ventricular aneurysm have been discussed.

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Streptococcal Pharyngitis Diagnosed by Throat Cultures in an Internal Medicine Practice

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This is a report of a 5-year experience with streptococcal upper respiratory infection in the private practice of general internal medicine. Epidemiologic studies have been done on military recruits,[1,2] sick and well, in pediatric clinics,[3-5] hospitals,[6,7] schools,[8-12] hospital outpatient departments,[13,14] and in private general practice and pediatricians offices,[15,16] but to our knowledge this is the first time such a study has been done in a private adult practice. A corollary purpose is to show that such clinical studies may be done in routine office practice and can make practice more stimulating and perhaps yield important data.

Method

Our patients were drawn mostly from the Washington, D.C. suburbs of Bowie, Maryland, and surrounding area. The index patients were all over 10 years of age. The patients could be classified as middle or upper middle in socioeconomic stratum. For the most part they had uncrowded, hygienic homes, schools, and public facilities.

Cultures were done on 3,653 patients who complained of sore throat, had pharyngitis by physical examination, had fever or cervical lymphadenopathy without other apparent cause, or had signs or symptoms suggestive of acute rheumatic fever. We strongly encouraged obtaining cultures in household contacts.

Swabs were made of the posterior pharynx and tonsillar areas and innoculated directly on blood-agar plates. A Bacitracin disc was placed on the area of thick smear, and plates were incubated at 37°C for 24 hours. They were considered positive for group-A beta hemolytic streptococci if opalescent colonies produced clear hemolysis and if the growth of these colonies was inhibited by the Bacitracin disc.[17] If there was doubt, the organisms were re-plated. We did not attempt to determine the Lancefield type of the streptococci so isolated.

Results

The results for the five year period from January 1, 1966 through December 31, 1970 are noted in tables 1 and 2.

Eight % of all persons cultured were found to have group-A beta hemolytic streptococci. Of patients meeting the criteria indicated above, 7.5% were found to have positive cultures (the index case). Positive cultures were found in 10.8% of those suspected of possibly harboring these bacteria because they were household contacts.

In households having an index patient 21.2% had one or more people who harbored the beta hemolytic streptococcus.

All of the index patients and contacts positive for beta hemolytic streptococci were treated with recommended dosage of schedules of penicillin, or erythromycin if penicillin allergy was suspected.[18] Among these 337 there was no instance of acute rheumatic fever or glomerulonephritis. During the same period we observed three new cases of acute rheumatic fever but no new cases of acute glomerulonephritis. All occurred in patients whom we had not recently seen for upper respiratory infections. Among the 274 patients with primary positive cultures we were able to obtain cultures of family contacts in 184 households, or 68%.

The incidence of infection was highest in the winter and early spring months. There is a rough correlation between the incidence of infection in our studied population and the incidence of positive contacts. The number of positive contacts in each month is too small to allow statistical comparison.

Discussion

The incidence of 'strep' throats among our primary suspects was 7.5%. This is toward the low side of the range of incidence found by other investigators and is a reflection of our patient population and our selection of patients to be cultured. Our index

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patients were all above the age of 10. The age of greatest susceptibility is age 6-12,[19] and most studies with a high incidence of infection have been done in grade school children.[2,3] We used very liberal criteria in deciding which patients were to be cultured. Other studies in adults cultured were only the clinically sicker patients in military hospital and public hospital emergency-room patients. Ours was a retrospective study of a population presenting itself in need of medical care. In a routine survey of cultures in a population of young asymptomatic military recruits in a non-epidemic situation, the incidence of 'strep' was 2.6% in over 12,000 cultures.[2] Since the populations and circumstances differ, one cannot actually infer that these asymptomatic recruits could act as controls in our study done some 10 years later. One can only speculate about the significance of the difference incidences in symptomatic and asymptomatic adults.

Twenty-one% of the households of patients with a positive culture had at least 1 member of that household with a positive culture.

Only 10.8% of the total number of contacts cultured harbored the beta hemolytic streptococcus. This again is toward the low side of the range found by other investigators, possibly because our population was adult, heterogenous, and mostly in a non-epidemic situation.

Of interest is the fact that on a purely voluntary basis, with families having to come to our office, we were able to obtain cultures from 68% of the households of our index patients. This was despite the cost to the patient. ($3.00 per culture, including adequate penicillin to treat infections.)

Seasonally the greatest incidence of infection was in winter and spring. In those months in which the incidence of 'strep' infection was high there was also a high incidence of positive cultures among the contact families. This suggests the possibility that epidemic strains are more contagious and virulent than endemic. Since streptococcal typing was not done we can only conjecture about the epidemiologic relationship between the patient and the family member with a positive culture. Indeed, there was no way of knowing whether the index patient had introduced the organism in the household or whether he was infected by the household member with the positive culture. Other studies indicated that the place of residence (barrack, home, etc.) is the commonest place of infection.[3,20] Differentiation between carrier and victim has proved difficult in all studies.

TABLE I: Results of Throat Cultures in Symptomatic

Year	Total Number of Cultures	Total Number of Positives	Total Percentage of Positives	Number Primary (Index) Cultures	Number Primary (Index) Positives	Percentage of Primary (Index) Positives
1966	417	38	9.1	362	28	7.8
1967	658	48	7.1	589	39	6.6
1968	772	56	7.25	696	44	6.3
1969	1029	98	9.5	839	83	9.9
1970	1354	97	7.2	1169	80	6.8
Total	4230	337	8.0	3653	274	7.5

TABLE 2: Results of Throat Cultures Contact Household Persons

Year	Number Contact Households Cultured	Number Contact Households Positive	Percentage Households Positive	Number Contact Individuals Cultured	Number Contact Individuals Positive	Percentage Contact Individuals Positive
1966	19	6	31.0	55	10	18
1967	24	4	16.7	71	9	12.7
1968	22	5	23.0	76	12	15.9
1969	64	13	20.4	190	15	7.9
1970	55	11	20.0	185	17	9.2
Total	184	39	21.2	577	63	10.8

It has once again been demonstrated by the absence of acute rheumatic fever and acute glomerulonephritis in our treated patients that prompt, adequate treatment of streptococcal sore throats leads to effective prevention of these illnesses. Penicillin reactions were unusual, and none was severe enough to require hospitalization.

Conclusion

Streptococcal infection is commonly found in the adult private practice. It may be simply, effectively and economically diagnosed and treated in the average physician's office. It is useful to culture family contacts. It is feasible to do simple studies in the office during routine practice. These may be more relevant to the practicing physician than studies done under other circumstances and can be of interest to the clinician and the medical community and of benefit to the patient.

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Muscle Damage and Acute Renal Failure Associated with Heroin Use*

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The widely disseminated scourge of drug abuse, specifically heroin addiction, has created unique and often frustrating dilemmas for the medical profession. The physician is particularly concerned with sequelae which are potentially reversible. The case report which follows describes a recently observed complication of heroin use, muscle damage with subsequent myoglobinuria and transient acute renal failure. This patient also exhibited chest findings consistent with heroin pulmonary edema.

Report of Case

I. C., a 26-year-old black man, came to the George Washington University Medical Center Emergency Room complaining of the sudden onset of muscle cramps of both lower extremities and dark urine. He was a frequent user of intravenous heroin; the most recent injection had been administered the previous evening. A history of loss of consciousness was equivocal, but he denied injury to his legs or symptoms of renal disease. On admission the blood pressure was 120/70, the pulse rate 80 and regular, and the temperature 36.8°C. The patient was not in acute distress. Head, eyes, including funduscopic exam, ears, nose and throat were normal. There were bilateral rhonchi posteriorly. The heart and abdomen were normal. Scars from needle tracts were apparent in the antecubital fossae. The muscles of the anterior compartment and flexors of the lower extremities were exquisitely tender below the knees. The calves were enlarged, and the dorsalis pedis pulses were 1+ and equal. There was decreased perception to pin prick symmetrically below the knee, but vibratory and position sense were intact. Initial chest X-ray films (Fig. 1A) revealed a diffuse bilateral pneumonic infiltrate, which cleared after four days (Fig. 1B). An ECG revealed peaked T-waves compatible with hyperkalemia but was otherwise normal.

Laboratory data on admission were: hematocrit 51 gm%, hemoglobin 17 gm, white blood cells 17,000/mm^3 with 74 segmented forms, 7 band forms, 11 lymphocytes, and 2 monocytes; urine dark, tea-colored, strongly benzidine-positive, with granular casts but no red blood cells on microscopic examination; specific gravity 1.015, pH 5.0, protein 2+, glucose and acetone negative; reticulocyte count 2%; electrophoresis, normal hemoglobin; electrolytes normal except for a serum potassium of 6.6 mEq/L. Chemistries are presented in figure 2: initial values SGOT* 925 (normal 10-50), LDH* 1100 (normal 90-200), CPK 313 (normal up to 20 sigma units), aldolase 290 (normal 3-8 S-L units), BUN* 22 mg/100 ml.

On the evening of admission the patient developed a decreased urine output of 260 ml over an eight hour period. Intravenous fluids, mannitol

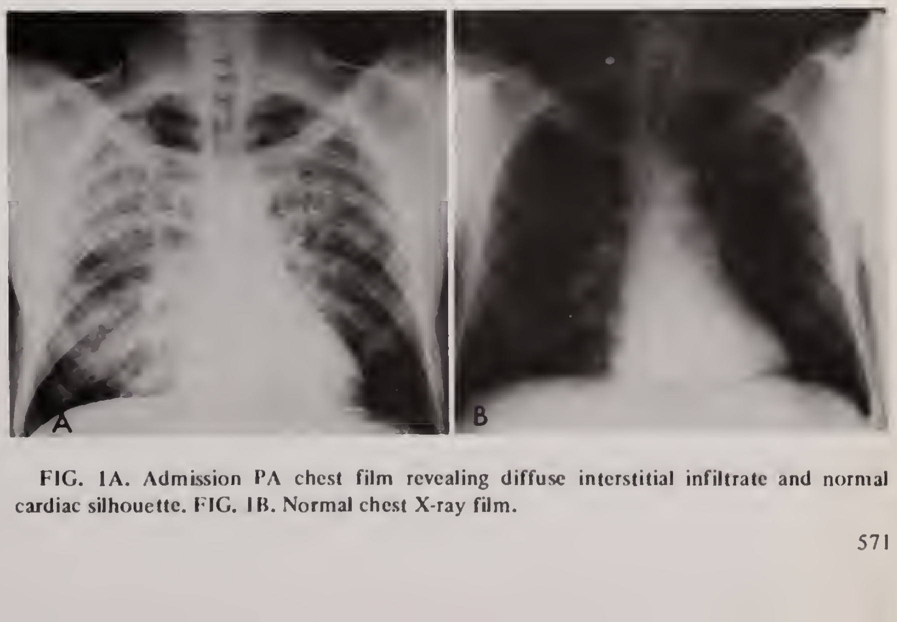

FIG. 1A. Admission PA chest film revealing diffuse interstitial infiltrate and normal cardiac silhouette. FIG. 1B. Normal chest X-ray film.

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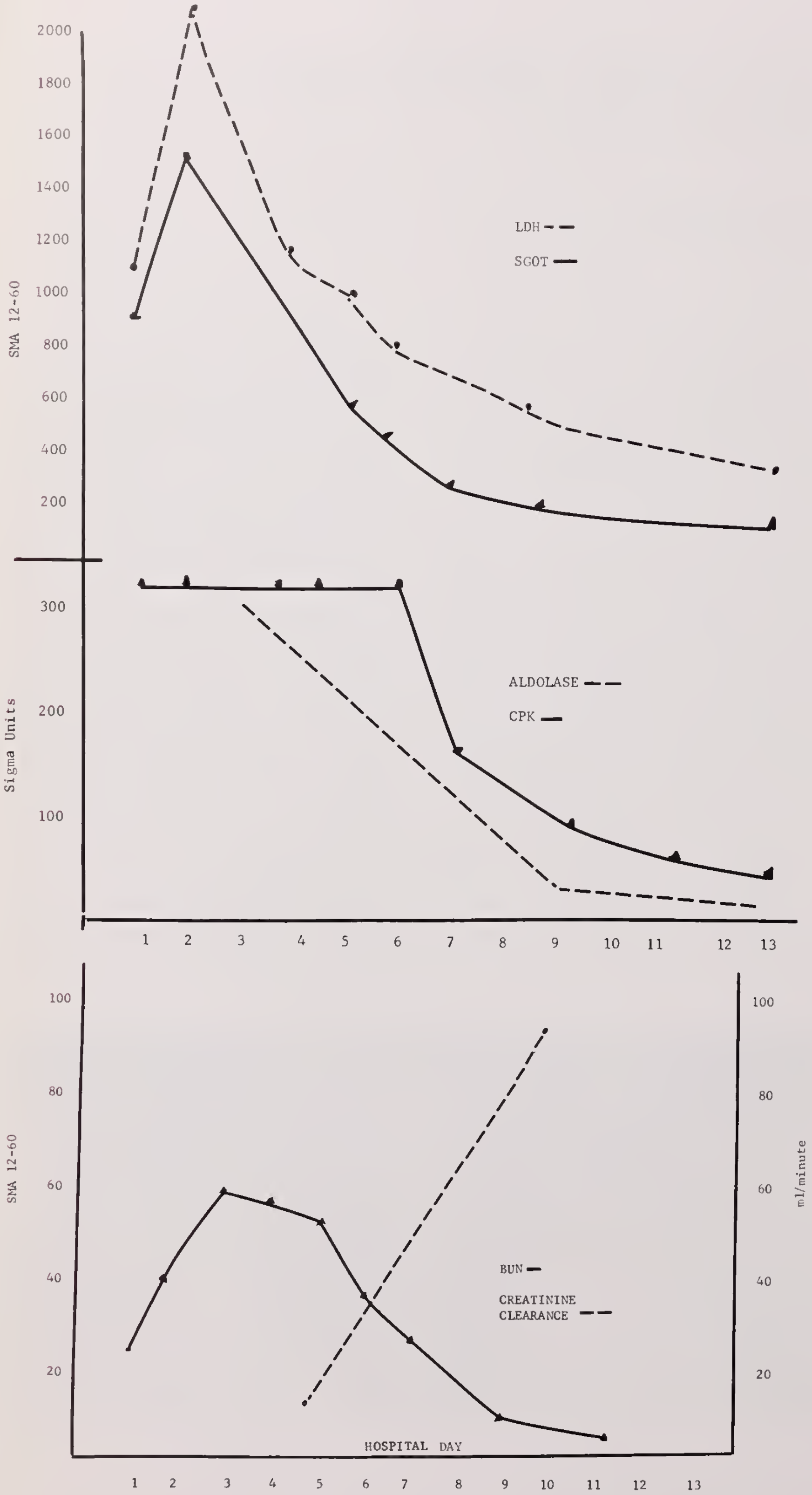

FIG. 2. Serum enzymes and renal function studies.

and lasix were administered, and a prompt diuresis ensued and was maintained at 2500-4900 ml/24 hours for one week. The urine became clear within 24 hours. On the third hospital day the BUN rose to 56 mg/100 ml, with serum creatinine 5.4 mg/100 ml, and creatinine clearance 16.9 ml/minute. At this time, it was noted that the blood pressures were in the range of 160/120 mm Hg.

The patient was treated with Methyldopa (Aldomet)®. The calf pain gradually subsided, and the enzymes returned to normal values over the next five days. Serum potassium values also reverted to normal. The patient was discharged after two weeks complaining only of slight calf tenderness. The blood pressure was normal, and antihypertensive medication was discontinued.

Special studies. An electromyogram was performed on the gastrocnemius muscles which revealed motor units of low voltage and short duration, and increased resting fibrillation potentials. These findings were interpreted as myopathic signs indicative of muscle damage. Another electromyogram ten weeks after discharge was completely normal. Light microscopy and muscle enzyme histochemistry studies of the involved gastrocnemius muscles obtained by muscle biopsy were normal. This was attributed to sampling error. Electron microscopy showed subsarcolemmal cylindrically arranged filaments thought to represent actin filaments (Fig. 3). A renal biopsy performed on the eighth hospital day revealed no pathologic changes (Fig. 4).

Discussion

Various medical complications attributed to the use of diacetylmorphine, coincident with the increase in numbers of drug users, have been reported in recent years. The pulmonary congestion noted in this case, which spontaneously resolved, has been seen with heroin use and attributed to either hypersensitivity[1] or increased pulmonary capillary permeability secondary to hypoxia.[2] However, current extensive reviews[3,4] do not mention the complication of rhabdomyolysis in heroin users, a condition recently described by Richter.[5]

The etiologies of myoglobinuria are diverse and include hereditary defects in muscle glycolysis and ischemia secondary to vascular occlusion. Various pharmacologic agents have been implicated in muscle damage. Licorice[6] and Amphotericin B[7] have been reported to induce hypokalemia with subsequent myopathy. Muscle necrosis following the use of succinylcholine and halothane anesthesia has been reported.[8] More commonly, a spectrum of muscle dysfunction including acute myolysis has been documented in alcoholics.[9] The mechanism of acute rhabdomyolysis in heroin users remains obscure. Some authors have postulated a "crush-like" syndrome[10,11] with limb compression as the mechanism of muscle necrosis. A hypothesis suggested by Richter[12] involves heroin or the heroin-adulterant mixture functioning as an exogenous antigen to skeletal muscle and thereby initiating an acute hypersensitivity reaction or a direct toxic effect on skeletal muscle.

The precipitation of protein and pigment in the renal tubules due to the release of large amounts of myoglobin is the usual mechanism attributed to the development of acute tubular necrosis (myoglobinuric nephrosis) seen in cases of extensive muscle damage. In-vitro studies[13] have demonstrated a direct toxic effect of heme pigments, including myoglobin, on renal tubular cells which may play a role in the development of acute renal failure. Diuretic treatment and alkalinization of the urine are effective if instituted early. However, in severe cases hemodialysis is required. The release of myoglobin may also have been responsible for the initial hyperkalemia observed in this patient, both directly and by inducing renal failure.[14]

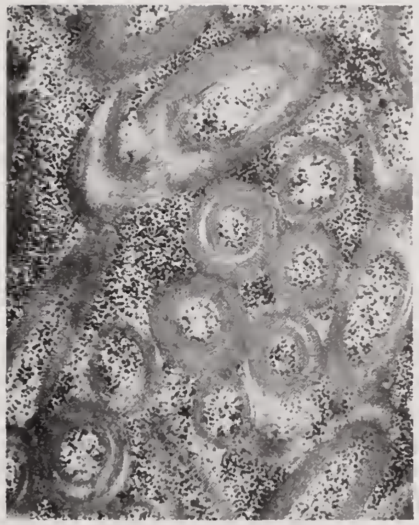

FIG. 3. Electron micrograph of gastrocnemius muscle revealing cyclindrically arranged filaments in the subsarcolemmal space. Preparation and interpretation, courtesy: Edward H. Bossen, M.D., Major, UC, USA; Armed Forces Institute of Pathology, Washington, D.C.

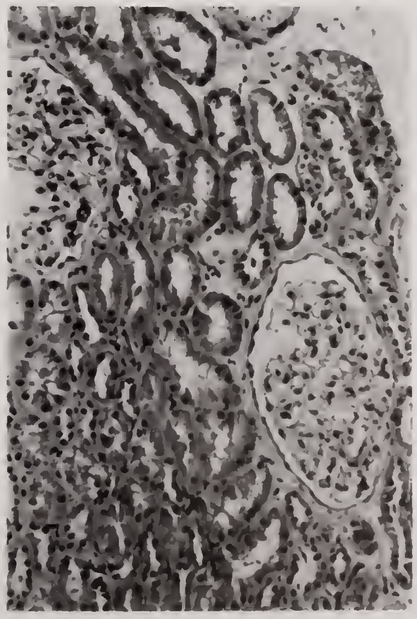

FIG. 4. Renal biopsy revealing normal histologic appearance.

Rather than advocate one of the several proposed pathophysiologic mechanisms for muscle damage in heroin users, we would stress the importance of early recognition of acute myolysis in drug abusers having severe muscle pain, and prompt treatment to avoid the morbidity and mortality associated with acute renal failure.

Summary

A patient has been presented with a history of intravenous heroin use, muscle pain, dark urine, and physical and roentgenologic findings consistent with heroin-induced pulmonary edema. He subsequently developed acute renal failure which was successfully treated with diuretics. Special studies were performed on biopsy specimens from the involved gastrocnemius muscles and revealed normal muscle enzyme histochemistry and normal histology by light microscopy. Electron microscopy showed abnormal collections of filaments, probably actin, but was otherwise non-specific. An electromyogram was abnormal, revealing myopathic changes in the affected muscles. Possible mechanisms which might account for the muscle damage seen in heroin users include "crush syndrome," a direct toxic effect, or a hypersensitivity reaction due to either heroin or the heroin-adulterant combination. Early recognition of this syndrome allows the prompt institution of therapy for acute renal failure.

Acknowledgments

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A Profile of the Graduates of Five U.S. Medical Schools

THOMAS M. PEERY, M.D.*

A 1968 statistical study, which seems to have been largely overlooked,† makes it possible for each American medical school to analyze certain characteristics of its own graduates. With the information contained in "Medical School Alumni, 1967," a school can evaluate the effects of institutional practices, policies and programs. Are admissions practices producing the desired results? Are educational programs effective and in proper balance? Have curriculum experiments produced the anticipated career motivation in the graduates? Partial answers to such questions can be derived from a carefully analysis of the "track record" of a school's graduates as revealed in this AMA source book.

Perhaps even more interesting, it is now possible to compare the graduates of any one American school with those of any other. State legislators may find such information of value as they consider the cost effectiveness of dollars spent in support of medical education.

"Medical School Alumni, 1967" is a paperback book in the Special Statistical Series published by the Department of Survey Research of the American Medical Association. Priced at $4.00 per copy, it can be obtained from the Order Department, American Medical Association, 535 North Dearborn Street, Chicago, Illinois 60610.

This source book is formidable; it is apt to turn one off before he appreciates the gold mine of information that is available between its covers. Its seemingly endless tables (table 1 runs along for 90 pages!) utilize unaccustomed abbreviations and column after column of figures to express the facts. Seldom is the presentation in exactly the form to suit the researcher's own special interests, but by careful reclustering of the figures much valuable information can be gleaned.

The data contained in "Medical School Alumni, 1967" have been derived from the files of the AMA Physician Records Service. The total physician population of the United States as of December 31, 1967 was studied in relation to the medical schools from which the physicians had graduated. As of that date there were 311,143 physicians known to be living in the United States. Of that number it was necessary to exclude 1660 because data were incomplete. The remaining 309,483 have been studied in considerable detail. Of the total, 81.69% were graduates of the 84‡ "active" U.S. medical schools, 1.2% were graduates of "extinct" U.S. medical schools, 2.1% were graduates of Canadian medical schools, and 15.0% were graduates of "foreign" medical schools. Of the total 46,347 graduates of foreign schools recorded at the end of 1967, 67.8% were graduated since 1949.

By a comparison of data in "Medical School Alumni-1967" with earlier published data,[1] two interesting observations are possible:

1. The U.S. physician population has increased by 28.4% from mid 1959 to the end of 1967. The increase in overall population of the U.S. during this same period was about 13%.

2. A very significant portion of this increase in physicians is a result of the accelerated entry of graduates of foreign schools. There were 31,193 more foreign physicians recorded in the U.S. in 1967 than in 1959, an increase of 205.8%.

Objectives of this Study

My study of "Medical School Alumni, 1967" was undertaken in order to obtain a profile of the alumni of The George Washington University School of Medicine. It was immediately apparent, however, that data relative to GW would have little meaning without reference to data from other schools. However, to compare GW's statistics to those of all U.S. medical schools would not permit evaluation of the effects of certain geographic, social and cultural similarities and/or differences. It was decided therefore to compare the total pool of GW's medical graduates with the total pool of medical graduates of four other schools. For this comparison the following medical schools were used: (1) Georgetown University School of Medicine, GW's sister school in the District of Columbia; (2) Medical College of Virginia (now known as the Health Sciences Division of Virginia Commonwealth University), a nearby state school of roughly comparable size; (3) University of Pennsylvania School of Medicine, and (4) Harvard Medical School, two Eastern schools with enviable reputations for excellence.

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† According to Science Citation Index, "Medical School Alumni, 1967" has been cited only twice since its publication (through 1971 listing).

‡ Not included, since they had no graduates as of December 31, 1967, are data on the 17 "developing medical schools" in the U.S.

A second objective of this study was to see if there were any significant changes in the characteristics of GW's alumni over the years, and if so, to seek the reasons for the changes. Certain tables in "Medical School Alumni, 1967" divide each school's graduates who were living on December 31, 1967 into 5-year groups according to the year of graduation, beginning with those graduated 1915-19. In this study, these 5-year groups have been reclustered into groups as follows: those graduated prior to 1929, those graduated 1930-1949, and those graduated 1950-1967. In "Medical School Alumni, 1967" we are cautioned against using these time-interval tables to detect trends, since large numbers of the earlier groups were dead before the remaining members of the groups could be studied, survivors may have moved to retirement locations, etc. Certainly the small size of the surviving members of the first group in table 2, i.e., 339, argues against its use for comparison. The second group – 1930 to 1949 – includes the war-accelerated classes; their career choices and other characteristics may be somewhat atypical. The third group – 1950 to 1967 – includes five classes, 1963-1967, many of whose members were still in internship, residency training or military service at the time the data were gathered; their specialty choices, location, and other data are not really suitable for comparison with the earlier, more settled classes. Nevertheless, the data are used, although with certain reservations.

Specialty Choices of the Graduates of Five U.S. Medical Schools

In table 1 the five medical schools are compared with regard to the distribution of their graduates among the various specialties. "Medical School Alumni, 1967" uses the list of 34 AMA recognized specialities, one of which is General Practice, for its tabulations. For our purposes (Table 1) I have combined six of the AMA's 34 with their parent specialties. Thus, Child Psychiatry is merged with Psychiatry, Diagnostic Roentgenology and Therapeutic Radiology are combined with Radiology, Forensic Pathology is included with Pathology, and Pediatric Allergy and Pediatric Cardiology with Pediatrics. While it would seem that 34 specialties should be enough, some physicians indicated that they were practicing a specialty other than those in the official list; these are clustered in the tables as "Unrecognized Specialties." There is an additional category, "Unspecified Specialty." It is not clear just what is meant by this category. Presumably it includes those physicians who did not check General Practice or any of the specified specialties and did not list an unrecognized specialty. It is likely that it includes a number of retired physicians, as is noted later. The data shown in tables 1 and 2 are derived from the physicians' own statements rather than evidence of certification by the various specialty boards.

Note that, of George Washington's (GW's) 3159 living graduates at the time of tabulation (first column of table 1) 623 (19.7%) indicated their specialty as General Practice. The comparable figure for Georgetown (GT) is 22%, Medical College of Virginia (MCV) 29.1%, Pennsylvania (Penn) 16.1%, and Harvard 6.5%.

For most of the specialties listed in table 1, GW occupies approximately a median position in comparison with the four other schools studied, as we did with regard to General Practice. Among these five schools, GW has the highest proportion of Obstetricians-Gynecologists, Pathologists, and Radiologists, and the lowest proportion of Pediatricians, but the differences are not very great. Harvard's low frequency of General Practice is naturally reflected as a higher incidence in almost every one of the specialties. Harvard's high percentage of surgeons, especially general surgeons, is noteworthy.

Trends in Specialty Choices of GW Medical Alumni

In Table 2, GW's 3159 medical alumni as of December 31, 1967 are divided into three columns according to their years of graduation in order to see if significant changes in specialty preference have occurred. In this table, only the major specialty categories of table 1 are used. Thus, all of the "Medical Specialties" listed in detail in table 1 are grouped together in table 2. Similarly the "Surgical Specialties" and "Other" categories are not subdivided in table 2.

Probably the most striking differences in percentage in each category between the time-interval groups of GW alumni shown in table 2 are (1) the marked decline in the "Unspecified" category between the first and second groups, and the rise between

TABLE I: Specialty Choices of the Graduates of Five U.S. Medical Schools
Based on Stated Specialties of Living Graduates as of December 31, 1967
(Derived from Table 1, "Medical School Alumni–1967", an AMA Publication)

	Gw	Gt	Mcv	Penn	Harvard
General Practice	623 (19.7%)	825 (22.0%)	1017 (29.1%)	958 (16.1%)	403 (6.5%)
Medical Specialties	571 (18.1%)	739 (19.7%)	533 (15.3%)	1143 (19.2%)	1629 (26.5%)
Internal Medicine	517	659	475	1013	1493
Allergy	10	8	5	25	11
Cardiovascular Diseases	24	35	24	54	81
Gastroenterology	9	14	10	23	23
Pulmonary Diseases	11	23	19	28	21
Surgical Specialties	516 (16.3%)	651 (17.3%)	509 (14.6%)	984 (16.6%)	1557 (25.3%)
General Surgery	298	412	313	621	1083
Neurologic Surgery	22	18	15	44	82
Orthopedic Surgery	93	100	86	157	170
Plastic Surgery	17	25	10	30	42
Colon and Rectal Surgery	7	3	9	7	10
Thoracic Surgery	21	19	15	33	68
Urology	58	74	61	92	102
Obstetrics & Gynecology	269 (8.5%)	282 (7.5%)	218 (6.2%)	392 (6.6%)	215 (3.5%)
Psychiatry (incl. Child Psych.)	195 (6.2%)	205 (5.5%)	130 (3.7%)	382 (6.4%)	452 (7.3%)
Pediatrics (incl. subspecialties)	146 (4.6%)	251 (6.7%)	194 (5.6%)	332 (5.6%)	395 (6.4%)
Radiology	139 (4.3%)	128 (3.4%)	124 (3.6%)	244 (4.1%)	172 (2.8%)
Ophthalmology	109 (3.5%)	113 (3.0%)	118 (3.4%)	217 (3.7%)	155 (2.5%)
Pathology	85 (2.7%)	73 (1.9%)	68 (1.9%)	152 (2.6%)	154 (2.5%)
Anesthesiology	72 (2.3%)	96 (2.6%)	71 (2.0%)	118 (2.0%)	59 (1.0%)
Other	218 (6.9%)	237 (6.3%)	277 (7.9%)	504 (8.5%)	481 (7.8%)
Aviation Medicine	12	5	12	14	13
Dermatology	37	45	42	87	49
Neurology	21	24	26	79	87
Occupational Medicine	20	21	29	51	30
Otolaryngology	57	57	68	97	52
Physical Med. & Rehab.	14	14	13	16	6
Preventive Med.	11	12	22	21	37
Public Health	14	11	36	39	41
Unrecognized Specialties	32	48	29	100	166
Unspecified Specialties	216 (6.8%)	155 (4.1%)	232 (6.6%)	517 (8.7%)	482 (7.8%)
Total	3159 (100%)	3755 (100%)	3491 (100%)	5943 (100%)	6154 (100%)

the second and third groups, (2) the decline in the category "General Practice," (3) the decline in the "Others" category, and (4) the slight but definite increase in almost all other categories.

It is quite clear that the large number (89) in the "Unspecified" category in the first group is due to the fact that the "Unspecified" category consists almost exclusively of retired physicians. This is confirmed by reference to table 4 in which the number in the "Inactive" category in the first group is seen to be 87. In the third group in table 2, the "Unspecified" category includes 76 physicians who were graduated 1965-67 and who, presumably, were undecided as to their career choices at the time of the tabulation on December 31, 1967. Since it is reasonable to assume that the inactive and undecided physicians comprizing the "Unspecified" category in all three groups represent the same distribution among specialties as the active group, the "Unspecified" group is not counted in the base (100%) used for calculating percentages for the other categories.

With this manipulation, the number of physicians in "General Practice" shows a steady decline: 34.8% in the earliest group, 26.0% in the 1930-1949 group, and 14.7% in the 1950-1967 group. The "Others" category also shows a significant decline. Reference to the original table from which these figures were derived shows that the

TABLE 2: Trends in Specialty Choices of GW Medical Alumni
Based on Stated Specialties of Living Graduates as of December 31, 1967
(Derived from Table 4, "Medical School Alumni–1967", an AMA Publication)

	? to 1929	1930-1949	1950-1967
General Practice	87 (34.8%)	321 (26.0%)	215 (14.7%)
Medical Specialties	36 (14.4%)	210 (17.0%)	325 (22.3%)
Surgical Specialties	32 (12.8%)	198 (16.0%)	286 (19.6%)
Obstetrics and Gynecology	17 (6.8%)	114 (9.2%)	138 (9.5%)
Psychiatry	10 (4.0%)	82 (6.6%)	103 (7.0%)
Pediatrics	12 (4.8%)	60 (4.9%)	74 (5.1%)
Radiology	9 (3.6%)	44 (3.6%)	86 (5.9%)
Ophthalmology	8 (3.2%)	38 (3.1%)	63 (4.3%)
Pathology	4 (1.6%)	33 (2.7%)	48 (3.3%)
Anesthesiology	4 (1.6%)	33 (2.7%)	35 (2.4%)
Others	31 (12.4%)	102 (8.3%)	85 (5.8%)
(Unspecified)	(89)	(31)	(96)
Base for Calculating %	250 (100%)	1235 (100%)	1458 (100%)
Totals	339	1266	1554

difference in the "Others" category is chiefly due to a decline in Otolaryngology from 4.8% in the first group to 1.6% in the 1950-67 group. An equally striking decline in Otolaryngology occurred in the graduates of the four reference schools in this study. Dermatology, Occupational Medicine and Public Health, all in the "Others" category, also showed a decline. All categories except "General Practice" and "Others" have increased in popularity among GW alumni in recent years.

"Major Professional Activities" of Graduates of Five U.S. Medical Schools

In table 3 the five medical schools are compared as to the ways in which their graduates practice, i.e., group practice, other private practice, etc. In order to avoid confusion between physicians in training and physicians practicing from a hospital base, all interns, residents and fellows have been excluded from the tabulations in table 3. Thus the totals in table 3 are different from those in table 1.

As seen in this table, George Washington's medical alumni compare fairly closely to graduates of Georgetown

TABLE 3: "Major Professional Activities" of Graduates of Five U.S. Medical Schools*
as of December 31, 1967
(Derived from Table 1 and Appendix V, "Medical School Alumni – 1967", an AMA Publication)

	GW	GT	MCV	Penn	Harvard
Patient Care	2446 (88.8%)	3010 (91.6%)	2852 (90.1%)	4450 (82.7%)	4090 (73.6%)
Group Practice†	295 (10.7%)	210 (6.4%)	361 (11.4%)	599 (11.1%)	656 (11.8%)
Other Private Practice	1751 (63.6%)	2342 (71.3%)	2108 (66.6%)	3229 (60.0%)	2711 (48.8%)
Hospital-Based Practice	400 (14.5%)	458 (13.9%)	383 (12.1%)	622 (11.6%)	723 (13.0%)
Other Professional Activity	179 (6.5%)	196 (5.9%)	160 (5.1%)	544 (10.1%)	1116 (20.1%)
Medical School Faculty	77 (2.8%)	89 (2.7%)	85 (2.7%)	313 (5.8%)	660 (11.9%)
Administration	70 (2.5%)	67 (2.0%)	44 (1.4%)	99 (1.8%)	147 (2.6%)
Research	32 (1.2%)	40 (1.2%)	31 (1.0%)	132 (2.5%)	309 (5.6%)
Inactive	126 (4.6%)	80 (2.4%)	153 (4.8%)	388 (7.2%)	352 (6.3%)
Totals*	2751 (100%)	3286 (100%)	3165 (100%)	5382 (100%)	5558 (100%)

* Interns, Residents and Fellows are excluded from these calculations.

† From Appendix V, "Medical School Alumni – 1967".

and the Medical College of Virginia in their commitment to Patient Care, whereas significantly larger numbers of the graduates of Pennsylvania and Harvard are committed to medical education and research. Of GW's alumni, 10.7% are in group practice, a figure significantly higher than that for Georgetown.

Trends in "Major Professional Activities" of GW Medical Alumni

In table 4 we can see certain trends in the way GW's medical graduates practice their profession. As in the calculations for table 2, the "Inactive" category has been excluded from the base used in calculating the percentages.

The great majority of GW's alumni in each group are involved in patient care. However, a significant shift is evident away from "Private Practice" toward "Hospital-Based Practice." A small increase is noted in the number of GW alumni in recent classes who are engaged in full-time medical school teaching and research, but it is questionable whether the shift is significant. Unfortunately, it is not possible to determine whether or not there is an increased participation in group practice among GW's alumni; this information cannot be derived from the AMA sourcebook.

TABLE 4: Trends in "Major Professional Activities" of GW Medical Alumni,*
Based on Living Graduates as of December 31, 1967
(Derived from Table 2, "Medical Alumni – 1967", an AMA Publication)

	? to 1929		1930-1949		1950-1967	
Patient Care	239	(94.8%)	1153	(93.4%)	1054	(92.6%)
Private Practice†	224	(88.9%)	1051	(85.1%)	771	(67.7%)
Hospital Based Practice	15	(6.0%)	102	(8.3%)	283	(24.9%)
Other Professional Activity	13	(5.2%)	82	(6.6%)	84	(7.4%)
Medical School Faculty	3	(1.2%)	36	(2.9%)	38	(3.3%)
Administration	10	(4.0%)	41	(3.3%)	19	(1.7%)
Research	0		5	(0.4%)	27	(2.4%)
(Inactive)	(87)		(26)		(13)	
Base for Calculating %	252	(100%)	1235	(100%)	1138	(100%)
Totals	339		1261		1151	

* Interns, Residents and Fellows have been excluded from these calculations.

† Data regarding group practice are not available by year of graduation.

Geographic Distribution of Graduates of Five U.S. Medical Schools

In table 5 the geographic distribution of the graduates of the five medical schools is shown as of the date of inquiry (December 31, 1967). George Washington, Pennsylvania and Harvard (and 20 other U.S. medical schools not shown in this table) had alumni in each of the 50 states and in the District of Columbia.

It is evident that GW's medical alumni, as well as the alumni of the four other schools studied, tend to locate in the more densely populated states of the Atlantic Seaboard in which all five of the schools are located. Of greater interest is the fact that a larger proportion of GW's alumni is located in the Southwestern and Mountain States than is the case with any of the 4 other schools studied. GW also has the largest percentage of its alumni located outside the United States.

The single jurisdiction having the most George Washington medical alumni is the District of Columbia (464); California is a close second (424), Maryland is third (320), New York is fourth (304), and Virginia is fifth (194). Georgetown has its largest concentration of medical alumni in New York (711); D.C. is second (419), New Jersey third (402), Pennsylvania fourth (342), and Maryland fifth (275). Medical College of Virginia's graduates have located, in order, in Virginia (1439), North Carolina (351), West Virginia (308), New York (131), and Florida (131). Pennsylvania's alumni are concentrated in Pennsylvania (2258), California (393), New Jersey (346), New York (337), and North Carolina (286). Harvard's medical alumni are located in Massachusetts (1606), New York (675), California (624), Maryland (241), and Connecticut (219).

Additional geographic data concerning the medical alumni of these five schools are shown in table 7. There it can be noted (line 5) that Georgetown and George Washington have the smallest proportion of their alumni practicing in the state where the medical school is situated, and also the smallest proportion in states contiguous to the medical school state (line (6)); in both instances this is probably due to the fact that the District of Columbia, in which both GW and GT are located, is very small in both area and population, as compared to the states of the other medical schools. The Medical College of Virginia has the largest proportion of its alumni settled in the state where the school is located (line 5). This may be related to admission policies of this State school; all of the other schools in this tabulation are private. The graduates of all five schools tend to locate in major metropolitan areas (line 4) having a population of 50,000 or more. This tendency is least evident in the graduates of the Medical College of Virginia (69.9%) and most evident in the graduates of Georgetown (90.3%).

TABLE 5: Geographic Distribution of Graduates of Five U.S. Medical Schools
as of December 31, 1967
(Derived from Table 7, "Medical School Alumni-1967", an AMA Publication)

	GW	GT	MCV	Penn	Harvard
Northeastern States					
Maine	9	20	4	19	85
New Hampshire	3	12	6	23	95
Vermont	3	2	1	10	31
Massachusetts	56	209	24	158	1606
Rhode Island	6	85	5	12	97
Connecticut	45	173	30	90	219
New York	304	711	131	337	675
Total, Northeast	426 (13.5%)	1212 (32.3%)	201 (5.8%)	649 (10.9%)	2808 (45.6%)
Mideastern States + D.C.					
Pennsylvania	148	342	79	2258	176
New Jersey	116	402	49	346	103
Ohio	87	77	88	174	217
Delaware	10	17	8	60	10
Maryland	320	275	80	142	241
District of Columbia	464	419	42	52	82
Virginia	194	160	1439	83	72
West Virginia	23	9	308	38	21
Total, Mideast	1362 (43.1%)	1701 (45.3%)	2093 (60.0%)	3153 (53.1%)	922 (15.0%)
Southeastern States					
North Carolina	52	33	351	286	143
Kentucky	17	14	23	35	45
Tennessee	18	8	57	45	58
South Carolina	10	16	52	31	11
Georgia	33	23	60	39	71
Florida	110	109	131	189	136
Alabama	23	19	20	50	45
Mississippi	5	4	11	30	19
Total, Southeast	258 (8.5%)	226 (6.0%)	705 (20.2%)	705 (11.9%)	528 (8.6%)
Great Lakes States					
Illinois	39	38	18	81	114
Indiana	14	19	18	20	36
Michigan	43	53	27	87	97
Wisconsin	34	13	11	70	80
Total, Great Lakes	130 (4.1%)	123 (3.3%)	74 (2.1%)	258 (4.3%)	327 (5.3%)
North Central States					
Minnesota	26	20	15	56	94
North Dakota	2	4	1	16	6
South Dakota	4	3	2	5	3
Iowa	13	2	7	16	16
Nebraska	1	5	2	12	14
Total, North Central	46 (1.5%)	34 (0.9%)	27 (0.8%)	105 (1.8%)	133 (2.2%)

TABLE 5–*Continued*

	GW	GT	MCV	Penn	Harvard
South Central States					
Missouri	15	19	13	50	88
Kansas	9	3	7	19	25
Arkansas	3	0	7	6	6
Oklahoma	13	6	6	16	24
Louisiana	18	10	9	30	18
Texas	59	38	70	113	115
Total, South Central	117 (3.7%)	76 (2.0%)	112 (3.2%)	234 (3.9%)	276 (4.5%)
Mountain States					
Montana	7	5	5	11	11
Idaho	24	3	1	12	13
Wyoming	5	2	0	4	5
Colorado	22	15	8	44	73
Utah	68	5	4	58	33
New Mexico	13	4	7	17	29
Total, Mountain States	139 (4.4%)	34 (0.9%)	25 (0.7%)	146 (2.5%)	164 (2.7%)
Northwestern States					
Alaska	4	0	3	9	8
Washington	62	15	13	79	126
Oregon	15	2	7	24	59
Total, Northwest	81 (2.6%)	17 (0.5%)	23 (0.7%)	112 (1.9%)	193 (3.1%)
Southwestern States					
Nevada	14	2	4	9	5
California	424	209	108	393	624
Arizona	29	11	19	37	29
Hawaii	7	3	4	11	24
Total, Southwest	474 (15.0%)	225 (6.0%)	135 (3.9%)	450 (7.6%)	682 (11.1%)
Outside USA					
Puerto Rico	29	35	38	15	15
Virgin Islands	0	0	0	3	0
Canal Zone	4	1	2	1	2
Foreign Countries	27	28	12	51	54
Military Overseas	56	43	44	61	50
Total, Outside USA	116 (3.7%)	107 (2.8%)	96 (2.7%)	131 (2.2%)	121 (2.0%)
Grand Total	3159 (100%)	3755 (100%)	3491 (100%)	5943 (100%)	6154 (100%)

Trends in Geographic Distribution of GW Medical Alumni

In table 6, GW's Medical Alumni are again divided into three groups according to the years of graduation, and geographic location of the alumni in these groups is noted. The designations of regions in table 6 are the same as in table 5, and the states contained in each region are the same. The column headed "?-1929" includes 87 inactive physicians whose locations may well represent retirement rather than practice. The column headed 1950-1967 includes 403 physicians who are still in internship, residency or fellowship training; a significant number of this

TABLE 6: **Trends in Geographic Distribution of GW Medical Alumni**
(Derived from table 7, "Medical Alumni – 1967", an AMA Publication)

	? to 1929	1930-1949	1950-1967
Northeastern States	76 (23.1%)	158 (11.9%)	186 (12.2%)
Mideastern States	170 (51.7%)	592 (44.5%)	600 (39.2%)
Southeastern States	32 (9.7%)	117 (8.8%)	119 (7.8%)
Great Lakes States	5 (1.5%)	47 (3.5%)	78 (5.1%)
North Central States	0 (0%)	14 (1.1%)	32 (2.1%)
South Central States	4 (1.2%)	52 (3.9%)	61 (4.0%)
Mountain States	5 (1.5%)	47 (3.5%)	87 (5.7%)
Northwestern States	6 (1.8%)	33 (2.5%)	42 (2.7%)
Southwestern States	31 (9.4%)	265 (19.9%)	274 (17.9%)
Outside USA	0 (0%)	5 (0.4%)	51 (3.3%)
Totals	329 (100%)	1330 (100%)	1530 (100%)

group can be expected to move again before settling down.

It can be noted that 75% of GW's medical alumni who graduated prior to 1930 and who were living on December 31, 1967 were located in the Northeastern and Mideastern States. Graduates after 1930 tend to be more widely scattered, especially to the Southwestern States.

Miscellaneous Characteristics of Five U.S. Medical Schools

In table 7 the alumni and faculty of the five medical schools are compared with regard to a number of different parameters recorded in various tables and appendices of the source book, "Medical Alumni-1967."

In line 1 it can be noted that George Washington has the highest proportion (6.5%) of women graduates of the five schools studied; the source book shows that there were only 21 active U.S. medical schools (out of 87) that had a higher percentage of women graduates.

Line 2 shows the overall percentage of alumni of each school that were certified by one or more of the American Specialty Boards. GW's alumni are in a median position in this regard, ranking above Georgetown and the Medical College of Virginia but below Pennsylvania and Harvard. These figures can be compared with the specialty choices as given in table 1.

In line 3 it can be noted that GW has a larger proportion of its medical alumni in federal employ than do any of the other schools listed here. The mean for all U.S. medical schools in the source book is 8.9%. Fifteen medical schools have a greater proportion of their graduates serving as federal employees. Most of these 15 are recently organized schools, many of whose graduates were in military service at the time the tabulation was taken.

Geographic information given in lines 4, 5 and 6 were discussed earlier in relation to table 5.

The record regarding medical school faculty reveals very significant information. Of the 5 schools compared, GW had the lowest ratio of full-time faculty per 100 medical students in each of the 3 categories tabulated: full-time M.D. basic science faculty (line 7), full-time M.D. clinical faculty

TABLE 7: MISCELLANEOUS CHARACTERISTICS OF FIVE U.S. MEDICAL SCHOOLS
(Derived from "Medical Alumni – 1967", an AMA Publication)

	GW	GT	MCV	Penn	Harvard
Data Regarding Medical School Graduates					
(1) Male Graduates, as % of Total Living Graduates (Appendix I)	93.5	97.9	94.1	96.0	97.7
(2) Specialty Board Certified, as % of Total Living Graduates (Table 6)	34.2	31.2	29.9	46.9	57.5
(3) Graduates Federally Employed, as % of Total Living Graduates (Appendix IV)	13.1	10.8	10.0	8.0	9.0
(4) % Graduates Practicing in Major Metropolitan areas (50,000) (App. VII)	86.1	90.3	69.9	86.5	89.4
(5) % Graduates Practicing in State where Medical School is (App. VI)	14.7	11.2	41.2	38.0	26.1
(6) % Graduates Practicing in States Contiguous to Medical School State (App. VI)	16.3	11.6	24.7	18.5	18.2
Data Regarding Medical School Faculty					
(7) "Full-time" MD Basic Science Faculty per 100 registered medical students*	3.5	5.8	6.1	6.6	20.9
(8) "Full-time" MD Clinical Faculty per 100 registered medical students*	24.2	35.6	35.2	39.0	55.2
(9) Total "Full-time" Faculty per 100 registered medical students*	37.8	52.4	65.3	62.1	112.5

* Based on data for the school year 1966–67, Appendix X.

(line 8), and total full-time faculty (line 9). These ratios are based upon data for the school year 1966-67. Since that date GW has added a significant number of full-time faculty, so that perhaps the record would be different today.

Discussion

From the data in "Medical School Alumni-1967" we can conclude that the majority (88.8%) of GW's alumni are involved in patient care, and that they share the trend toward specialization which began in the 1930's and was accelerated after World War II. A large number of GW's graduates are employed in the federal services, larger than the mean of all U.S. medical schools. While a significant number of our alumni settle in the Mideastern States where the school is located, GW is truly a national school, whose alumni are to be found in every state of the Union, with a remarkable pocket of concentration in the Southwestern and Mountain States.

There are, obviously, certain limitations to the value of such a study. In the first place, it would be desirable to compare the findings of the present study with some earlier study, but this is possible only to a limited degree since the only earlier collection of comparable data was assembled somewhat differently. It is hoped that the AMA study will be repeated at regular intervals, perhaps every 10 years, to correspond with census data, so that the effects of changes in policies in the different medical schools can be noted.

It is suggested that other medical schools might use this unique source book to derive data regarding their alumni. The possibility of comparison of 1 school's alumni with another's, using the same parameters and degree of objectivity, is a particularly attractive feature.

References

1. Health Manpower Sourcebook, Section 11, Medical School Alumni. Public Health Service Publication No. 263, 1961.

Indications for Myocardial Revascularization

MICHAEL G. SEREMETIS, M.D.*

Surgery for coronary artery disease has come a long way and is becoming widely accepted as a mode of treatment in this field. Direct revascularization with either aortocoronary saphenous vein bypass or internal mammary-coronary anastomosis are presently the preferred procedures. Other operative approaches such as implantation of the internal mammary arteries (Vineberg) are applied less frequently and constitute a supplementary choice in certain cases. Certain complications of coronary artery disease such as ventricular aneurysm, ventricular septal defect or valvular insufficiency due to ruptured chordae tendinae are, of course, treated surgically and in these instances the procedures can be combined.

The goals of surgery for revascularizing the myocardium always have been the relief of incapacitating symptoms, mainly pain, the prevention of further damage to the cardiac muscle by ischemia and the improvement in the mortality rate of the disease. Protection from sudden death may also be included in the latter.

The development of practical technics for coronary arteriography by Sones in 1958 and later by Judkins was a milestone and opened the way for excellent objective evaluation of the coronary arteries and thus permitted the most accurate selection of patients for surgery. No patient is now accepted for revascularization unless he has a preoperative coronary arteriogram. The location and extent of the disease can be determined and the surgical approach tailored to the lesions present. From the experience of large centers it is now apparent that the majority of patients can be treated surgically, from the technical point, that is, a bypass graft can be connected successfully to distal coronary vessels even in the presence of extensive disease.

The selection of patients for coronary artery surgery has taken, however, a new direction with the evaluation of the patients already operated upon. It is clear that the patency rate of approximately 85% of grafts implanted two years ago or longer is very acceptable. The improvement, however, of the ventricular function values measured by objective methods shows much better results in the patient whose left ventricular myocardium is more or less unimpaired from the effects of ischemia, fibrosis or true infarcted areas. Patients already in heart failure do not show objective improvement. Whether they will show longer survival will have to wait the test of time. Emphasis is, therefore, placed upon the EARLY study and possible surgery of symptomatic patients before the myocardium suffers irreparable damage.

The subject of impending infarction is a very interesting one. An already large number of patients have been operated upon with excellent results. The definition of this condition is still based on clinical and laboratory observations, including serial electrocardiograms. It is possible to make a fairly accurate diagnosis this way but it would be ideal, however, if we had tests for objective assessment of the viability of myocardial cells.

Revascularization of the myocardium can be achieved with increasingly better results in acute coronary occlusions. This is a wide field presenting tremendous challenge. The study of the patient with an acute occlusion requires the use of circulatory assistance, a field that is still in the developing stage. The lack of tests to assess myocardial viability is more obvious in the patient with cardiogenic shock. Efforts to study and save these patients depend entirely on the condition of the muscle and its effectiveness as a pump. Coronary artery surgery in these patients may limit the extent of the infarcted area and may prevent irreversible changes. In addition infarctectomy for akinetic or dyskinetic myocardium may certainly improve left ventricular function in many patients.

In conclusion, the selection of patients for coronary artery surgery is based today on the feasibility of restoring adequate blood supply to ischemic areas *early* in the course of the disease and eventually should include all the acute coronary occlusions or impending infarctions as emergencies like acute arterial occlusions are treated elsewhere in the body. Further restoration of adequate left ventricular performance is possible with infarctectomy, combined with revascularization.

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Debt Collection

In our economy, credit is easily obtained but debt collection is often difficult to achieve. Assuming you have rendered medical services to a patient who has not paid any part of your bill, you may decide that you want to dun the patient for payment. As this point, you should be aware of the credit laws as they affect you, and of the problems that may arise in attempting to collect the debt in a lawful manner.

I. The Law on Extending Credit

The Federal Truth in Lending Act (the "TLA") and Regulation Z ("Reg. Z") of the Federal Reserve Board thereunder contain rules that apply in every jurisdiction. The TLA applies to all persons who in the ordinary course of business extend, offer to extend, arrange or offer to arrange for the extension of consumer credit. Reg. Z defines "consumer credit" as credit offered or extended to a natural person in which the money, property or service of the transaction is primarily for a personal, family or household purpose and for which either a *finance charge is or may be imposed* or which *pursuant to an agreement* is or may be *payable in more than four installments.*

The physician will be within the scope of the TLA if he imposes a finance charge or enters into a verbal or written agreement with a patient for payment in more than four installments. However, since Reg. Z uses the phrase "pursuant to an agreement" in its definition of consumer credit, the acceptance of installments from a patient, where there is no formal agreement, does not necessarily place the physician under the TLA or Reg. Z; this is because payment of less than the full amount is a unilateral act by the patient. So long as the physician bills the patient for the entire amount in each billing, the fact that the physician may accept installments in partial payment does not necessarily constitute an agreement to do so on his part. Furthermore, although the Federal Reserve Board recognizes that the sending of monthly statements requesting full payment does permit the patient to pay his bill in installments, Reg. Z will not apply as long as the physician has not agreed to accept more than four installments. Finally, where the physician treats a patient over a period of time and periodic payments for services are made as they are rendered, the TLA and Reg. Z will not apply.

The physician may avoid application of the TLA and Reg. Z by not imposing a finance charge *and* by not agreeing (either verbally or in writing) with his patient to accept payment of the physician's fee in more than four installments. If, however, the physician does impose a finance charge or agree to accept his fee in more than four installments, then he will be required to comply with the credit transaction disclosure requirements of the TLA. A brief disclosure will satisfy the TLA (fig. 1).

Reg. Z also provides that if under state law a class of credit transactions is subject to requirements substantially similar to those imposed under the Federal law, then the Federal Reserve Board will exempt such class of transactions in that state from the TLA.

The Virginia Consumer Credit Code is such a state law and it requires an interpretation similar to the TLA. Thus, where the Virginia physician imposes a finance charge or agrees with his patient to accept payment of his fee in more than four installments, the aforementioned TLA disclosure procedure will satisfy the state law as well.

The TLA and Reg. Z are applicable in Maryland and the District of Columbia since neither state has a law substantially similar to the TLA requirements.

II. The Law on Collecting Debts

Whether or not the physician successfully avoids the disclosure requirements of the credit laws, he will need to know how he may lawfully collect the debt. The TLA and Virginia Consumer Credit Code are laws requiring the disclosure of certain information in the extension of credit; such disclosure laws do not outline a course of conduct for the collection of a debt.

The District of Columbia, however, has such a statute dealing with debt collection in its recently enacted Consumer Credit Protection Act of 1971 (the "CCPA"). The CCPA applies, in part, to conduct and practices in connection with the "collection of obligations arising from

---

This disclosure is in compliance with the Federal Truth in Lending Act:

Dr. John Doe
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. . . .
(Patient's Name) (Patient's Address)

1. Cash Price (Medical Fee) ________________
Less: Cash Down Payment ________________
2. Unpaid Balance of Cash Price ________________
3. Amount Financed ________________
4. FINANCE CHARGE ________________
5. Total of Payments (3 + 4) ________________
6. Deferred Payment Price (1 + 4) ________________
7. ANNUAL PERCENTAGE RATE ________________

The "Total of Payments" shown above is payable to Dr. John Doe at the address shown above in . . . . . monthly installments of $ . . . . each, the first installment being payable . . . . , 19 . . . . . . , and all subsequent installments being due on the same day of each consecutive month until paid in full.

. . . .

. . . .
(Date) (Patient's Signature)

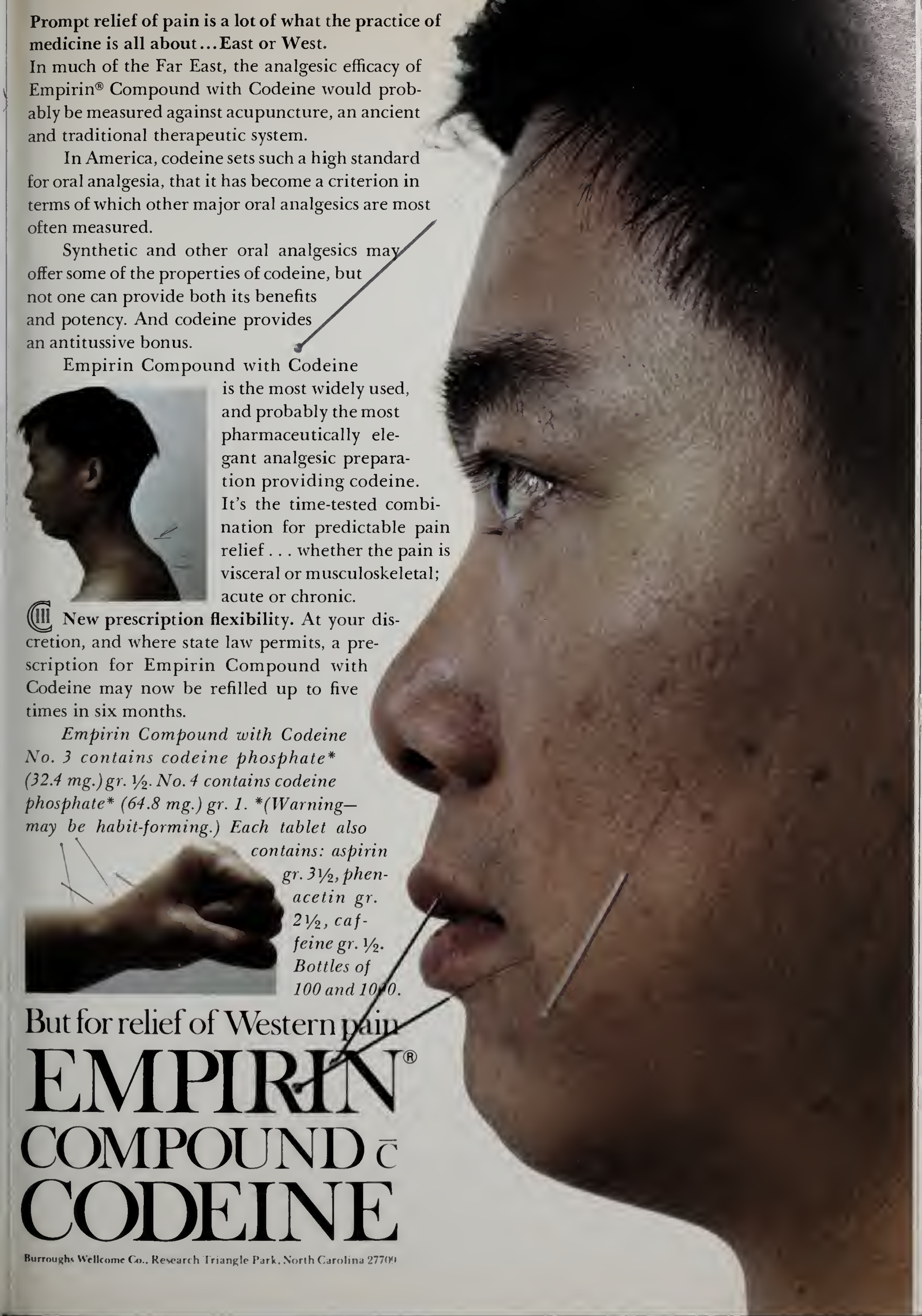
FIG. 1. Credit Transaction Disclosure

consumer credit sales". A "consumer credit sale" is defined as a sale of goods or services in which:

(a) Credit is granted by a person who regularly engages as a seller in credit transactions of the same kind;
(b) the buyer is a natural person;
(c) the goods or services are purchased primarily for a personal, family, [or] household. . .purpose;
(d) either the debt is payable in installments or a finance charge is made; *and*
(e) the amount financed does not exceed $25,000.

Since some provisions of the CCPA are a direct outgrowth of the TLA, it is probable that the CCPA will also apply to physicians. Note that by not imposing a finance charge *and* by not entering into a bilateral agreement (either verbal or written) for payment in installments, the physician in D.C. can avoid both the disclosure requirements of the TLA and the conduct requirements of the CCPA.

Assuming that the CCPA does apply to a physician, it becomes necessary to examine the statute. The CCPA provides that no debt collector shall collect or attempt to collect any money alleged to be due and owing by means of any threat or coercion. No debt collector shall *unreasonably* oppress, harass or abuse any person in connection with the collection of a claim, by the use of profane or obscene language that will *unreasonably* abuse the hearer or reader, or by the use of the telephone, without disclosure of the caller's identity, intending to harass or threaten the person called. Other provisions prohibit a debt collector from *unreasonably* publicizing false information relating to any debtor or alleged



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Warnings: Patients with severe cardiac disease should be given this medication with caution. Fever and possibly heat stroke may occur due to anhidrosis.

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Diarrhea in an ileostomy patient may indicate obstruction, and this possibility should be considered before administering Pro-Banthīne.

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Adverse Reactions: Varying degrees of drying of salivary secretions may occur as well as mydriasis and blurred vision. In addition the following adverse reactions have been reported: nervousness, drowsiness, dizziness, insomnia, headache, loss of the sense of taste, nausea, vomiting, constipation, impotence and allergic dermatitis.

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indebtedness, or from using fraudulent or deceptive representations, or unfair or unconscionable means to collect or attempt to collect any claim.

Unlike the District, neither Maryland nor Virginia has a statute which prohibits the harassment of a debtor by a creditor. There is case law in Virginia, however, that allows a debtor to collect damages from a creditor who illegally attempts to coerce payment of a debt by communications with the debtor's employer. In addition, a recent Maryland Court of Appeals decision allowed a debtor to recover $9,000 in damages for invasion of privacy by a creditor who continually harassed and threatened the debtor and her 11-year-old child by calling on the telephone over 200 times within a six-month period.

These cases, and decisions in other jurisdictions, indicate that the courts are concerned with the deliberate and persistent harassment of debtors. The use of insulting and outrageous language in oral or written communications over a substantial period of time, or the unreasonable interference with the debtor's business, occupation or employment, are courses of improper conduct which may subject the creditor to liability for damages. Statutes such as the District's CCPA are developing as a result of such cases. Therefore, whether he practices in Maryland, Virginia or the District, the physician is advised that when he attempts to collect on a delinquent account, he must do so within the guidelines of the CCPA and case law establishing standards of reasonableness for the debt collection.

III. Conclusion

When a physician extends credit to his patients in the regular course of his practice, he faces possible regulation under the credit laws. If the debt is payable in more than four installments or if a finance charge is made, the Federal Truth in Lending Act (or the Consumer Credit Code in Virginia) will require the full disclosure of the credit transaction to the patient.

Even if the physician avoids the disclosure requirements of the credit laws, he will find

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authority preventing him from attempting to collect the debt in an improper manner. The harassment statute in the District of Columbia, although not expressly applicable to physicians, provides a good framework for proper debt collection which the Virginia and Maryland practioner should also heed. The physician may dun his patient by calling and/or sending letters, provided that both are done in a courteous and professional manner over a reasonable period of time. If no response is forthcoming, the physician may turn his bills over to a collection agency, which, of course, is bound by the same collection standards.

Edited by Stefan F. Tucker of Arent, Fox, Kintner, Plotkin and Kahn, Washington, D.C.

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HELP WANTED
To Control Venereal Disease

Next month, area medical societies, hospitals, and private practitioners will join governmental agencies and local residents in a program to control the spread of venereal diseases in Greater Metropolitan Washington.

During the week of October 9, area-wide facilities will be available for diagnosis and treatment of venereal diseases. This will be preceded by a health education and information program to encourage utilization of medical facilities.

Venereal disease is on a rampage. It is one of the major public health problems in the country. In 1960, some 12,000 cases of Primary and Secondary Syphilis and almost a quarter million cases of Gonorrhea were reported. Within little more than a decade, the numbers have increased; more than 23,000 cases of Primary and Secondary Syphilis and more than 624,000 cases of Gonorrhea were reported during 1971.

And if these statistics are not disturbing enough, the federal government noted that there may be three to four times the number of reported venereal diseases that do not come to the attention of local and state health departments.

The statistics in the District of Columbia reflect those of the nation. In 1960, 472 cases of Primary and Secondary Syphilis and some 11,000 cases of Gonorrhea were reported. In 1971, 569 cases of Primary and Secondary Syphilis and more than 11,000 cases of Gonorrhea were reported. It is expected that these numbers will increase during 1972.

The District of Columbia has the fifth highest Infectious Syphilis rate and the seventh highest Gonorrhea rate among 160 cities and counties in the United States.

Our reporting system is one of the reasons the venereal disease rate in D.C. is high. Venereal disease specialists in the Community Health and Hospitals Administration look for, treat and report venereal disease contacts. When urban areas report low venereal disease rates, there may be some suspicion of their reporting system.

Nationally, the venereal disease rate for teen-agers and young adults has doubled during the years 1956 and 1971. It has been said that 20 per cent of all high school students contract venereal disease before they get a high school diploma. In the District of Columbia more than 60 per cent of the reported venereal disease cases are among those 15 to 24 years old.

One of the complications in venereal disease control is the nature of the disease. Many women have gonorrhea and may not know it. It has been estimated that 80 per cent of all women who have gonorrhea in the District of Columbia are asymptomatic and may not know that they have the disease. It is important that these women are found and receive a medical diagnosis and treatment.

Those in public health know that recognition of community health problems and provisions for medical care are important ingredients in solving these problems. Yet, important as they are, another phase is necessary if we are to reduce morbidity rates. People must know how to enter the health care system, and be motivated to take the necessary action. All too often, well intended community health programs miss their mark when these are not built into the program.

This is why we have developed a V.D. community health education and information program. Carefully planned, our objectives are to provide information about venereal diseases to community residents. We want people to know that venereal disease is preventable. We also want people to be able to recognize symptoms of venereal disease and take the necessary action for diagnosis and treatment early. During the week of October 9, we will encourage men and women to utilize medical resources, and special emphasis will be placed upon asymptomatic females.

Our strategy is community involvement. Through our committee structure, local residents are planning and helping to implement the program. In addition to a medical committee, spearheaded by representatives of the area medical societies, other committees including public information, school, community action, speakers, special events, and church have been organized in the District of Columbia. Participating jurisdictions in the Greater

Washington Metropolitan Area are encouraged to involve community residents similarly. These jurisdictions are Montgomery and Prince Georges counties in Maryland and Alexandria, Arlington, Fairfax, Prince William, and Loudoun counties in Virginia.

Area-wide, we will encourage patients to bring in their contacts for diagnosis and treatment. Physicians can assist in this important activity. They should encourage their patients to bring in contacts. Physicians should also report to the local health jurisdictions the information required by law concerning patients who are treated for venereal disease. Only with thorough field investigations can we ever hope to break the chain of infection.

Through the years, veneral disease organisms have become resistant to treatment. The recommended amount of penicillin to treat uncomplicated gonorrhea has increased 700 percent since 1948. About 25 years ago, 600,00 units were recommended; today 4.8 million units of aqueous procaine penicillin G are used.

Venereal disease is out of control. We can not tolerate this condition to continue. Now is the time to stop the spread of venereal disease. We need your cooperation, along with the assistance of every person in the community.

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book REVIEWS

Office Gynecology. ALVIN F. GOLDFARB, M.D. Price, $33.75. Baltimore: Williams and Wilkins, 1971.

Dr. Alvin F. Goldfarb has produced in three and one-half hours an excellent although cursory review of office gynecology as practiced today. The "book" consists of three audio cassettes and a supporting manual. It is an excellent review for both the newly graduated house officer as well as the established practitioner. The section on dysfunctional uterine bleeding was particularly well done. I would have liked to have heard more about cryosurgery as an office procedure. His delivery is both erudite and interesting and readily lends itself to being listened to while driving in one's car or sitting in one's study for closer attention. I would recommend it particularly for the resident preparing to go into practice, but also for the actively practicing gynecologist for the particular attention paid to details which one might not recall in the rush of a busy practice.

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Diabetes Mellitus: Diagnosis and Treatment. Vol. III. STEFAN S. FAJANS, M.D. AND KARL E. SUSSMAN, M.D., Co-editors. Price, $5.75. Pp. 430. New York: American Diabetes Association, 1971.

This third volume published by the American Diabetic Association is an over view of the common and complex disease, Diabetes Mellitus. It is appropriate that this volume should be published in the fiftieth anniversary year of the discovery of insulin. The book proposes to review the current progress in Diabetes as well as, to enlarge upon various topics introduced in the first two volumes. It is edited by Drs. Fajans and Sussman with chapters contributed by eighty authors, expert in particular areas of carbohydrate metabolism.

The book is divided into eight basic categories. These include: Basic Considerations, Diagnosis, Therapy, Problems in Management, Pregnancy and Diabetes, Emergencies, Long-term Problems, and Special Problems.

The most exciting and interesting sections of the book concern the work of the last five years dealing with the pathophysiology of diabetes. Prominent discussions include the role of Proinsulin, mechanism of insulin secretion and release and the seemingly important role of Glycogen in carbohydrate homeostasis. Discussion of the histology of the basement membrane in diabetes and its relation to insulin deficiency is well discussed. Many of the sections on diagnosis and therapy are simply text book reviews of standard thinking in these areas. Two chapters are devoted to the pros and cons of oral hypoglycemic therapy in diabetes in light of the UGDP study. Opthalmologic problems and possible therapy in diabetic retinopathy were especially well discussed in five chapters.

The editors as well as the numerous authors, present a comprehensive and clear picture of the present day knowledge of diabetes. This book is aimed at the physician particularly interested in the care of the diabetic patient. It can, however, serve the student or other interested physicians as a reference to investigate particular problems they may face in the area of carbohydrate metabolism. The book serves the very important function of compiling into one volume the current thoughts, controversies and new developments in the field of diabetes. It is recommended on that basis.

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vol. 41 no. 9 september 1972

Drug Design, Volume I. I. J. ARIENS, Editor. Price, $33.00. Pp. 581 with illustrations. New York: Academic Press, 1971.

This book will be of limited usefulness to the practicing physician. In the words of the editor it "is expected to be of particular interest to medicinal chemists, pharmacologists, biochemists, pharmacists, and, in general, scientists interested in the study of bioactive compounds."

The present volume is part of Volume 11 of a series of monographs on medicinal chemistry published by Academic Press from 1963 through 1971. Other volumes of the series cover more specific subjects such as Diuretics, Psychopharmacological Agents, Cardiac Stimulant Substances, and Oral Hypoglycaemic Agents. Two other parts of Drug Design are forthcoming. It would be less confusing if these separate books were designated Book I, II, etc. or Part I, II, etc. The term volume should be restricted to the separate monographs on other subjects in the same series.

It is suggested that the term drug design means "the effort to develop drugs on as rational a basis as possible." At the same time, the specific and complex titles of these articles resemble a "presentation in the field of science fiction." The chapters of the present volume are: A General Introduction to the Field of Drug Design, Quantitative Structure – Activity Relationships in Drug Design, Physiochemical Approaches to the Rational Development of New Drugs, A Molecular Orbital Approach to Quantitative Drug Design, Electronic Aspects of Drug Action, The Role of Biopharmaceutics in the Design of Drug Products, and Significance of Pharmacokinetics for Drug Design and the Planning of Dosage Regimens. Part of these articles were originally presented as lectures at the Symposium on Drug Design held at the Nijmegen University on July 8–10, 1969. A perusal of the book indicates that drug design is hardly a science fiction in spite of the fragmentary and incomplete knowledge of many aspects of the field. The various presentations vary in quality and complexity, but all contain worthwhile material with unusually complete documentation. A certain amount of overlapping is unavoidable.

Chapter 1, nearly half the book, by E. J. Ariens, contains much information frequently overlooked by clinicians. A review of the multiple and interrelated factors affecting drug action from dosage form, through pharmacokinetics, including drug-receptor interaction, to side effects is given. Discussion then follows of the procedures employed in drug design, past and present, with many well chosen and informative examples. Every biological action detected presents a potential lead. A natural product with some activity may lead to screening of several thousand compounds as in the case of the antimalarials related to quinine. Study of the side effects of various sulfonamides produced by molecular manipulation led to antidiabetic, diuretic, and hypotensive agents. The general purposes of molecular manipulation are outlined followed by brief discussion of the design of suitable forms for administration, dosage regimens and clinical evaluation. The concluding sections on structure and action, agonists and antagonists and submolecular approach to drug action relate primarily to mechanisms of drug action rather than drug design. The chapter as a whole presents an extensive review of the multifaceted and rapidly changing areas of research in the field of medicinal chemistry.

A. EARL VIVINO, M.D.

Central Nervous System Investigation with Radionuclides. ALBERT J. GILSON, M.D. AND WILLIAM M. SMOAK, III, M.D. Editors. Price, $27.50. Pp. 415 with illustrations. Springfield, Illinois: Charles C Thomas, 1971.

This text is a compilation of papers presented at the Second Annual Nuclear Medicine Seminar of the University of Miami held in March 1970. (The fact that it is an early 1970 work is carefully concealed by the publisher). Because it is impossible for all interested practitioners to attend every important symposium, it is important that papers from selected meetings be published; however, this is one seminar that should have been selected not to have been published. And this is unfortunate because the nuclear medicine literature still remains severely disseminated and this was an excellent opportunity to collate in one volume the current state of the art in terms of concept and practical applications of the uses of radiotracers for investigation of CNS diseases. Indeed, this was the intent of the editors but unhappily for us, they did not succeed. They tried, and the fact that they did not succeed was through no fault of theirs.

Specific criticisms are as follows: four of the 21 eminent Guest Faculty, 20 per cent, had already published the substance of their talks elsewhere and did not contribute to the book; nineteen of the 33 published chapters, 58 per cent, (some of whom had also published their contributions elsewhere) appear without a single reference; many of the chapters were obviously reworked from their oral delivery and specially prepared for this text, while others were just as obviously simply typed from the tape recordings of the presentations and were printed without critical review of the content. All of this resulted in some (few) papers being interesting and informative, while many were sketchy, superficial and trivial. Some were so general that they were vague and offered little in the way of practical guidance. Notable exceptions were Lassen's illuminating review of cerebral blood flow measurements in health and disease and Sweet's interesting documentation of the "Historical Development of Tracer Uses in CSF Dynamics". Dr. Sweet, who is Chief of Neurosurgery at Massachusetts General Hospital, refers to no less than 14 (that I could identify) separate published works, yet does not list a single bibliographic source. Several of the discussions were provoking, particularly the intercourse between a neuroradiologist, Dr. Fredie Gargano and a nuclear medicine physician, Dr. Henry Wagner, on how they would individually want to be worked up for an abrupt "vascular" accident, i.e., what tests they would want performed with implication of normal and abnormal results, the sequence of tests performed, and over what period of time interval.

In spite of the many shortcomings, there is more information than one finds in the CNS chapter in available nuclear medicine textbooks, and this makes this book useful as a temporary reference source. The only problem is that anyone actively involved in performing, interpreting, or using these nuclear medicine procedures, and who is only moderately "up" on the literature, will find precious little to help him.

A lot more could be said about this book, but too much has been written already. This book cannot be recommended for the personal library of practitioners of nuclear medicine, neurology, neurosurgery, or to house staff or medical students. In this time of properly structuring one's priorities, $27.50 can be better spent elsewhere.

RICHARD C. REBA, M.D.

Chest Medicine. P. S. SHANKAR. Price, RS 21.50. Pp. 239. Bombay: Current Technical Literature Co. Pvt. Ltd., 1971.

This is "a concise account of various diseases affecting the respiratory system for the medical students and practitioners" by a British trained Indian physician. It does not attempt to compete with the encyclopediac pulmonary texts which provide reference for the internists and pulmonary specialists, so the question is whether it is superior to internal medicine texts. The presentation is more detailed, but this is offset by the poor photographs, limited style, and frequent spelling and grammatical errors. For example, the statement that disodium chromoglycate in asthma "is suitable for those getting perineal attacks and not for those who get attacks occasionally" is, at least at first glance, confusing.

Because tuberculosis is the most important pulmonary disease in India, this section should be definitive and geographically appropriate. Concepts of treatment are good, but the statement that ethambutal and rifampicin "are not indicated in initial therapy" might be justified at present on a public health basis because of the cost, but not on the basis of effectiveness or toxicity. The statement that "radiological examination may be done at the end of six months and one year" after initiation of chemotherapy is different from current practice in the United States, and may reflect cost and limited facilities, but also makes us reconsider what really is essential. The presentation of intermittant chemotherapy and home care is appropriate, as is the endorsement of BCG.

This book has little utility in the United States except for the comparative medical aspect.

HAROLD M. SILVER, M.D.

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Reach to Recovery. TERESE LASSER AND WILLIAM KENDALL CLARKE. Price, $5.95. Pp. 158. New York: Simon and Schuster, 1972.

Although aimed at the post mastectomy woman, this book also should be read by surgeons and other physicians having contact with these patients. Terese Lasser had a mastectomy herself and one of the American Cancer Society's best programs, "Reach to Recovery" grew out of her experiences. The book is her biography but in reality only uses her story as a thinly disguised scaffolding on which to bring out the various problems of the mastectomy patient. And they are varied and real; such as bathing suits, being off balance, sexual adjustments, etc. Many of these a male doctor may never think of and his female patient may be too embarrassed to ask about. The book answers the many questions a postoperative patient would have and could be suggested to these women and their families shortly after surgery. It is easily readable, nontechnical and moderately priced. Many M.D.s would learn much from this thin volume which they could easily read in one night and thereafter keep in their offices.

ALLEN M. MONDZAC, M.D.

Quantitative Organ Visualization in Nuclear Medicine. PETER J. KENNY AND EDWARD M. SMITH, Editors. Price, $20.00. Pp. 910 with illustrations. Coral Gables, Florida: University of Miami Press, 1972.

The "Three P Concept" is mentioned about midway in this compilation of a 1971 Miami symposium. A London physicist's question about computer Processing for the Physician in Podunk evoked several opinions. One view favored a slow approach before getting involved in large data processing systems. Later, an American physicist noted that although clinical usefulness of processing techniques had not been demonstrated, this simply reflected the state of the art. At the final summation, an eminent American radiologist concluded we were at the same position that existed five years ago in the development of computerized radiation treatment planning.

The scope of this book is very broad. Some of the conferees felt much of the work presented was of a refresher course variety. Others, that it stressed highly technological material and not enough of practical application to routine clinical problems. These objections indicate precisely the contents of the book. There is thorough coverage of organ-imaging systems and each component is examined in detail with physical and mathematical exposition. The physician with no computer background may feel lost occasionally, even if he choses to skip over the mathematical sections. The state of the art and the directions it is taking—or may take—are well described. Analog and digital recording, on and off-line computer use, positron camera, optical filtering and holography are some of the subjects covered. There is an excellent section on camera calibration.

Quantitative organ visualization has already been used to measure thyroid function, calcium uptake in the skeletal system and the rate of red blood cell destruction by the spleen. As shorter-lived radionuclides become more available, dynamic function studies will increase in number and importance. It is in this field particularly that computer assistance will be most valuable. Whether non-university Nuclear Medicine facilities will within a five to ten year span have their own computers or be tied in by special telephone lines on a time-sharing basis will be decided by future developments in technology, cost and man-power. Closer liaison between clinicians and equipment manufacturers must obtain.

The book is a comprehensive presentation of the subject. It contains many figures, tables, and photographs. The typographical errors are minimal. This book will be of value primarily to Nuclear Medicine Departments in university hospitals and can be useful to other Nuclear Medicine physicians.

CORNELIUS F. MURPHY, M.D.

Frontiers in Neuroendocrinology 1971. LUCIANO MARTINI AND WILLIAM F. GANONG, Editors. Price, $25.00. Pp. 419 with illustrations. New York: Oxford University Press, 1971.

The physician actively engaged in neuroendocrinological research may find this frontier volume helpful. American and European investigators review from their own personal experience current areas of research activity in neuroendocrinology. This is the second volume of a series of biennial books intending to provide timely summaries of this field.

Anatomical aspects of the relationship among hormones, releasing factors, and the central nervous system receive some emphasis. The first chapter reviews the histology of the hypothalamic associations with the pituitary. Later chapters review research on the hypophyseal portal vasculature and the anatomical site of corticosteroid feedback control of ACTH secretion. Some obscure flames may be added to the fires of women's liberation by the declaration in the review chapter on sexual aspects of neuronal development that "the critical hormonal factor in normal physiological development in the presence or absence of a testicular product, presumably antigen." I personally was more interested in the review chapter on neuroendocrinological factors affecting sexual behavior. The authors noted a "marked similarity between the changes in ejaculatory activity of Rhesus monkeys and in the incidence of sexual intercourse in a human population," and speculated that "similar basic underlying neuroendocrinological mechanisms may be responsible for the behavioral changes in the two species." Included also in this review volume are chapters on the regulation of FSH, LH, and prolactin secretion. Recent work on the releasing factors is reviewed; some attention also is given to inhibiting factors released by the hypothalmus. The hormonal control mechanisms governing salt and water intake receive a lucid summary in another chapter.

As usual, the Oxford University Press has done a superb job with the printing and binding of this volume. I do quarrel with the inclusion of references in the body of the text. In one article I noted seven consecutive lines filled with the names of workers and the dates of their work in that particular field. Such footnoting in the body of the text always disrupts my train of thought and makes it difficult to follow the writing. I see no reason why footnotes can't be kept at the end of the chapter.

Clearly, the work reviewed in this volume is at least a decade, if not two, removed from clinical medicine. Nevertheless, any regular reader of *Scientific American* may profit from the lucid discussions of current problems in the field of neuroendocrinology.

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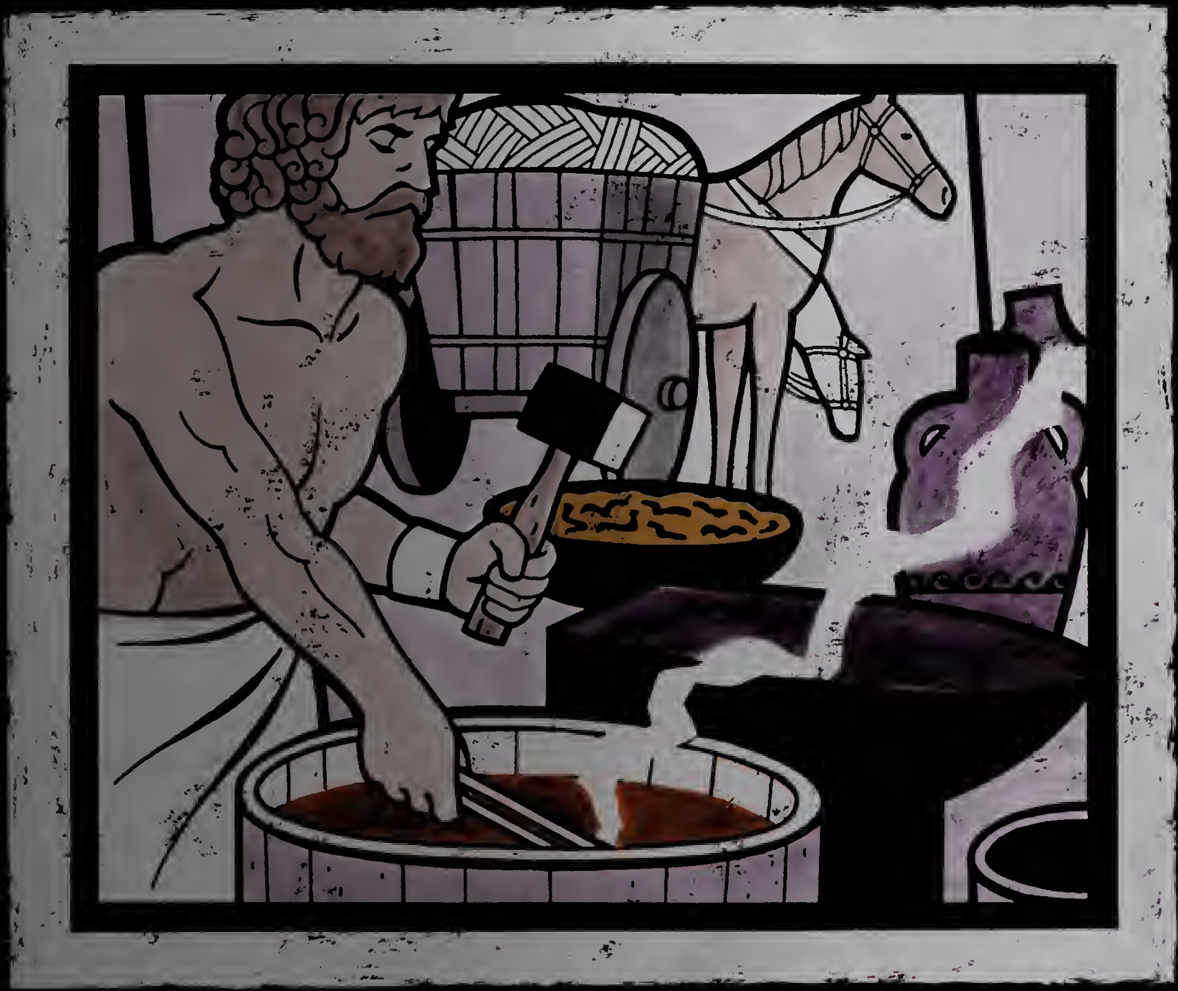

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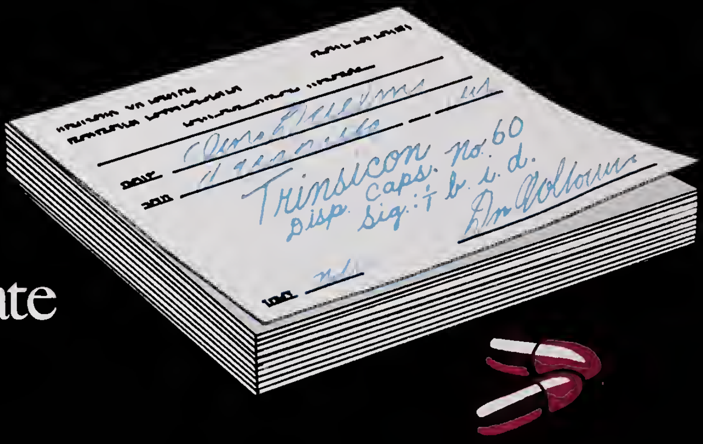

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Folic Acid	0.5 mg.

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Indications: Trinsicon is a multifactor preparation effective in the treatment of anemias that respond to oral hematinics, including pernicious anemia and other megaloblastic anemias and also iron-deficiency anemia. Therapeutic quantities of hematopoietic factors that are known to be important are present in the recommended daily dose.

Vitamin B_{12} with Intrinsic Factor—When secretion of intrinsic factor in gastric juice is inadequate or absent (e.g., in Addisonian pernicious anemia or after gastrectomy), vitamin B_{12} in physiological doses is absorbed poorly, if at all. The resulting deficiency of vitamin B_{12} leads to the clinical manifestations of pernicious anemia. Similar megaloblastic anemias may develop in fish tapeworm (*Diphyllobothrium latum*) infection or after a surgically created small-bowel blind loop; in these situations, treatment requires freeing the host of the parasites or bacteria which appear to compete for the available vitamin B_{12}. Strict vegetarianism and malabsorption syndromes may also lead to vitamin B_{12} deficiency. In the latter case, parenteral therapy, or oral therapy with so-called massive doses of vitamin B_{12}, may be necessary for adequate treatment of the patient.

Potency of intrinsic factor concentrates is determined physiologically, i.e., by their use in patients with pernicious anemia. The liver-stomach concentrate with intrinsic factor and the vitamin B_{12} contained in two Pulvules Trinsicon provide 1½ times the minimum amount of therapeutic agent which, when given daily in an uncomplicated case of pernicious anemia, will produce a satisfactory reticulocyte response and relief of anemia and symptoms.

Concentrates of intrinsic factor derived from hog gastric, pyloric, and duodenal mucosa have been used successfully in patients who lack intrinsic factor. For example, Fouts *et al.* maintained patients with pernicious anemia in clinical remission with oral therapy (liver extracts or intrinsic factor concentrate with vitamin B_{12}) for as long as twenty-nine years.

After total gastrectomy, Ficarra found multifactor preparations taken orally to be "just as effective in maintaining blood levels as any medication that has to be administered parenterally." His study was based on twenty-four patients who had survived for five years after total gastrectomy for cancer and who had been taking two Pulvules Trinsicon daily.

Folic Acid—Folic acid deficiency is the immediate cause of most, if not all, cases of nutritional megaloblastic anemia and of the megaloblastic anemias of pregnancy and infancy; usually, it is also at least partially responsible for the megaloblastic anemias of malabsorption syndromes, e.g., tropical and nontropical sprue.

It is apparent that in vitamin B_{12} deficiency (e.g., pernicious anemia), lack of this vitamin results in impaired utilization of folic acid. There are other evidences of the close folic acid-vitamin B_{12} interrelationship: (1) B_{12} influences the storage, absorption, and utilization of folic acid, and (2), as a deficiency of B_{12} progresses, the requirement for folic acid increases. However, folic acid does not change the requirement for vitamin B_{12}.

Iron—A very common anemia is that due to iron deficiency. In most cases, the response to iron salts is prompt, safe, and predictable. Within limits, the response is quicker and more certain to large doses of iron than to small doses.

Each Pulvule Trinsicon furnishes 110 mg. of elemental iron (as ferrous fumarate) to provide a maximum response.

Ascorbic Acid—Vitamin C plays a role in anemia therapy. It augments the conversion of folic acid to its active form, folinic acid. In addition, ascorbic acid promotes the reduction of ferric iron in food to the more readily absorbed ferrous form. Severe and prolonged vitamin C deficiency is associated with an anemia which is usually hypochromic but occasionally megaloblastic in type.

Contraindications and Precautions: Anemia is a manifestation that requires appropriate investigation to determine its cause or causes.

Folic acid *alone* is unwarranted in the treatment of pure vitamin-B_{12}-deficiency states, such as pernicious anemia. Indeed, the use of folic acid in large doses in pernicious anemia without adequate vitamin B_{12} may result in hematologic remission but neurological progression.

As with all preparations containing intrinsic factor, resistance may develop in some cases of pernicious anemia to the potentiation of absorption of physiological doses of vitamin B_{12}. If resistance occurs, parenteral therapy, or oral therapy with so-called massive doses of vitamin B_{12}, may be necessary for adequate treatment of the patient. No single regimen fits all cases, and the status of the patient observed in follow-up is the final criterion for adequacy of therapy. Periodic clinical and laboratory studies are considered essential and are recommended.

In extremely rare instances, skin rash suggesting allergy has been noted following the oral administration of liver-stomach material. Allergic sensitization has been reported following both oral and parenteral administration of folic acid.

Hemochromatosis and hemosiderosis are contraindications to iron therapy.

Adverse Reactions: In rare instances, iron in therapeutic doses produces gastrointestinal reactions, such as diarrhea or constipation. Reducing the dose and administering it with meals will minimize these effects in the iron-sensitive patient.

Dosage: One Pulvule twice a day. (Two Pulvules daily produce a standard response in the average uncomplicated case of pernicious anemia.)

How Supplied: Pulvules Trinsicon® (hematinic concentrate with intrinsic factor, Lilly), in bottles of 60 and 500 and in Identi-Dose® (unit dose medication, Lilly) in boxes of 100.

[090971]

Trinsicon®
Hematinic Concentrate with Intrinsic Factor
A Comprehensive Hematinic

Additional information available to the profession on request.

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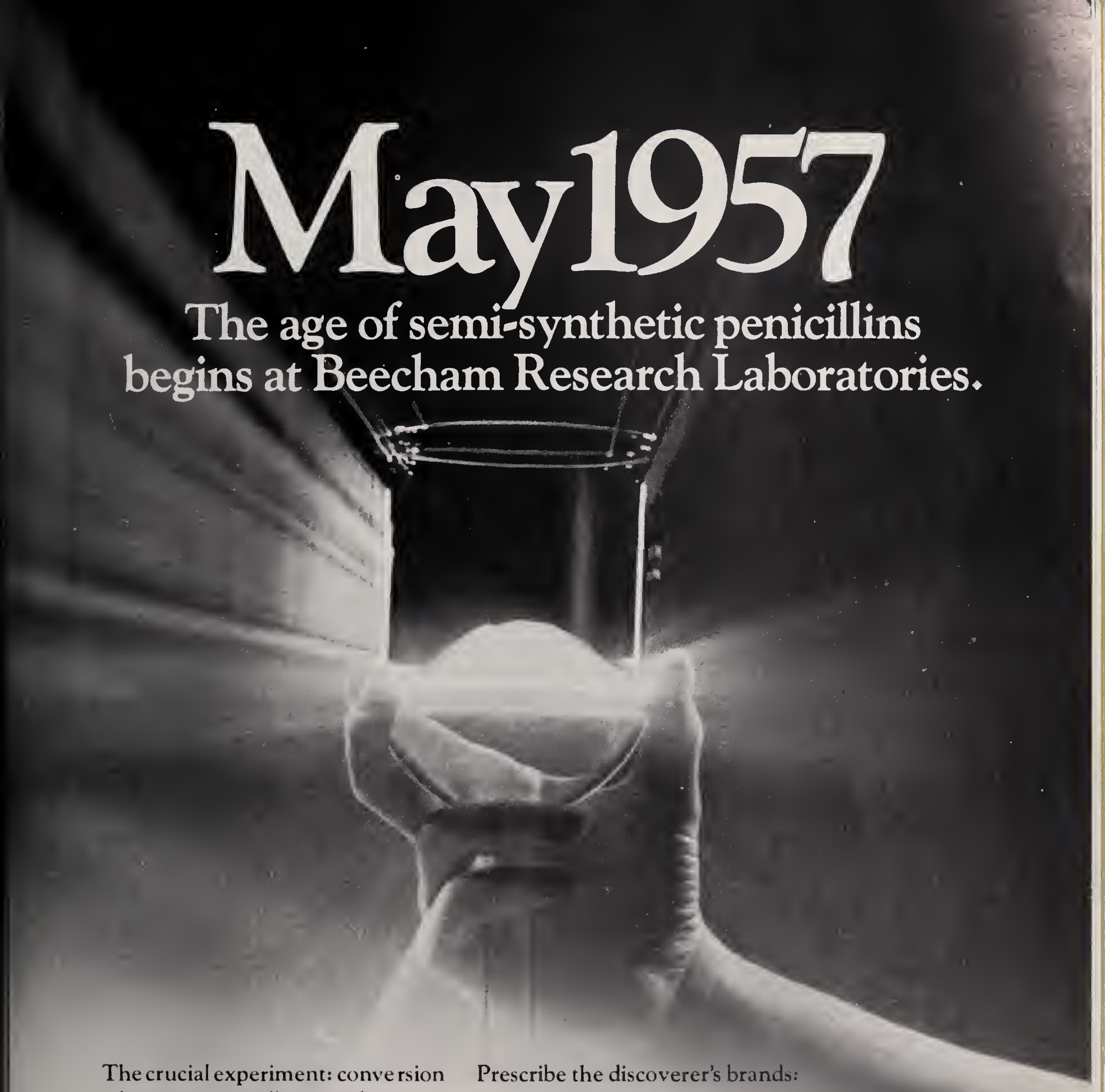
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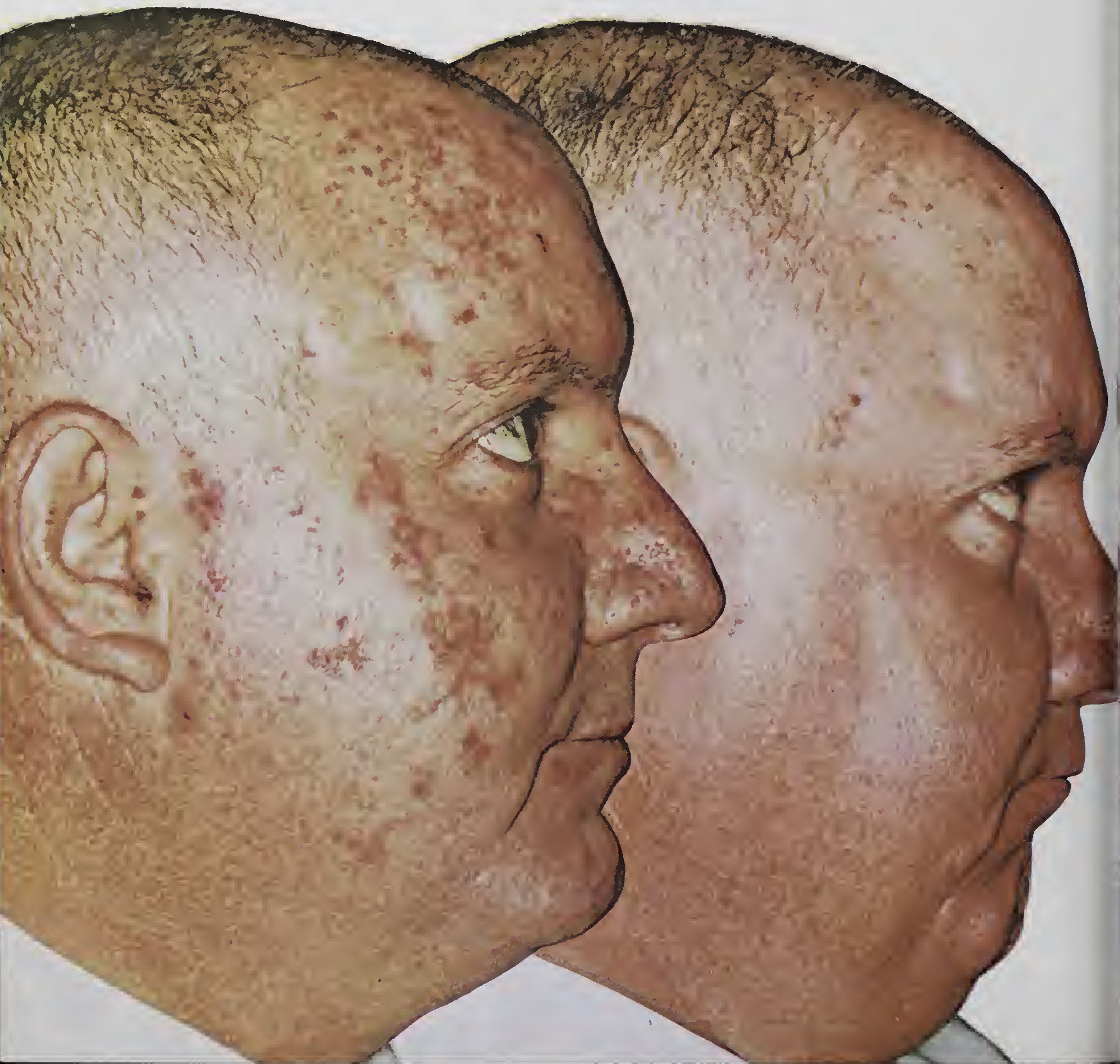
Call it what you will, it may be premalignant.

Before

3/29/67   Before therapy with 5%-FU cream. Patient P. T. shows a moderately severe solar keratotic involvement. Note residual scarring from the previous cryosurgical and electrosurgical procedures on forehead and ridge of nose adjacent to periauricular area.

After

6/12/67   Seven weeks after cessation of therapy. Reactions have subsided. Residual scarring is not seen except for that due to prior surgery. Inflammation has disappeared and face is clear of keratotic lesions.



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nd Efudex® (fluorouracil) 5% cream can resolve it.

Cll it actinic, solar or senile keratoses, ny regard it as "precancerous."[1,2]

ical fluorouracil, considered by some dermatologists to be a major
ance in the treatment of multiple solar keratoses,[3,4] offers the physi-
a relatively inexpensive alternative to cryosurgery, electrodesic-
on and cold knife surgery. Of the topical fluorouracils available, only
dex offers 2% and 5% solution and 5% cream formulations—formula-
s that have proved effective in the treatment of these mutliple lesions.

ual duration of therapy, 2 to 4 weeks.

lies showed that with the 2% and 5% Efudex preparations, the usual
tion of therapy was only 2 to 4 weeks.[5] Other studies with topical
ouracil revealed that when concentrations of less than 2% were
l, significant numbers of lesions recurred.[6]

eats the lesions you can't see, too.

nerous lesions, not apparent prior to 2% and 5% Efudex therapy,
ifested themselves by definite reactions, while intervening skin
ained relatively unaffected.[5] The early eradication of these subclini-
esions (which may otherwise have undergone further progression)
ably accounts for the reduced incidence of future solar keratoses in
ents treated with topical fluorouracil—especially with 5%
entrations.[6]

w to identify solar keratoses.

ically, the lesion—a flat or slightly elevated brown to red-brown
le—is dry, rough, adherent and sharply defined. Multiple lesions
the rule.

dictable therapeutic response.

response to a typical course of Efudex therapy is usually
acteristic and predictable. After 3 or 4 days of treatment, erythema
ns to appear in the area of keratoses. This is followed by a moderate
tense inflammatory response, scaling and occasionally moderate
erness or pain. The height of this response generally occurs two
s after the start of therapy and then begins to subside as treatment
pped. Within two weeks of discontinuing medication, the
mmation is usually gone. Lesions that do not respond should
opsied.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Multiple actinic or solar keratoses.

Contraindications: Patients with known hypersensitivity to any of its components.

Warnings: If occlusive dressing used, may increase inflammatory reactions in adjacent normal skin. Avoid prolonged exposure to ultraviolet rays. Safe use in pregnancy not established.

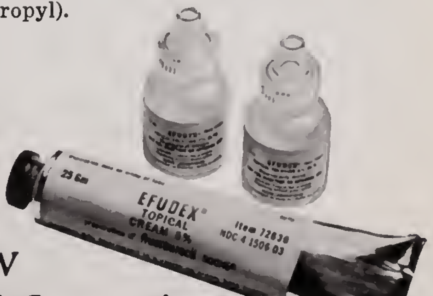
Precautions: If applied with fingers, wash hands immediately. Apply with care near eyes, nose and mouth. Lesions failing to respond or recurring should be biopsied.

Adverse Reactions: Local—pain, pruritus, hyperpigmentation and burning at application site most frequent; also dermatitis, scarring, soreness and tenderness. Also reported—insomnia, stomatitis, suppuration, scaling, swelling, irritability, medicinal taste, photosensitivity, lacrimation, leukocytosis, thrombocytopenia, toxic granulation and eosinophilia.

Dosage and Administration: Apply sufficient quantity to cover lesion twice daily with nonmetal applicator or suitable glove. Usual duration of therapy is 2 to 4 weeks.

How Supplied: Solution, 10-ml drop dispensers—containing 2% or 5% fluorouracil on a weight/weight basis, compounded with propylene glycol, tris(hydroxymethyl)aminomethane, hydroxypropyl cellulose, parabens (methyl and propyl) and disodium edetate.

Cream, 25-Gm tubes—containing 5% fluorouracil in a vanishing cream base consisting of white petrolatum, stearyl alcohol, propylene glycol, polysorbate 60 and parabens (methyl and propyl).



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ences: 1. Allen, A. C.: *The Skin, A Clinicopathological Treatise*, ed. 2, New York,
& Stratton, 1967, p. 842. 2. Dillaha, C. J.; Jansen, G. T., and Honeycutt, W. M.:
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3. Belisario, J. C.: *Cutis*, *6*:293, 1970. 4. Sams, W. M.: *Arch. Derm.*, *97*:14, 1968.
a on file, Hoffmann-La Roche Inc., Nutley, New Jersey. 6. Williams, A. C., and
E.: *Cancer*, *25*:450, 1970.

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Highly effective. Active against pinworm...and roundworm.

Non-staining. Doesn't stain teeth or oral mucosa on ingestion. Doesn't stain stools, clothing or linen.

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Well-tolerated. Based on pre-introductory studies.

Pleasant-tasting. Easy-to-take, caramel-flavored oral suspension.

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Antiminth (pyrantel pamoate) Oral Suspension

Actions. Antiminth (pyrantel pamoate) has demonstrated anthelmintic activity against *Enterobius vermicularis* (pinworm) and *Ascaris lumbricoides* (roundworm). The anthelmintic action is probably due to the neuromuscular blocking property of the drug.

Antiminth is partially absorbed after an oral dose. Plasma levels of unchanged drug are low. Peak levels (0.05-0.13 μg/ml) are reached in 1-3 hours. Quantities greater than 50% of administered drug are excreted in feces as the unchanged form, whereas only 7% or less of the dose is found in urine as the unchanged form of the drug and its metabolites.

Indications. For the treatment of ascariasis (roundworm infection) and enterobiasis (pinworm infection).

Warnings. *Usage in Pregnancy:* Reproduction studies have been performed in animals and there was no evidence of propensity for harm to the fetus. The relevance to the human is not known.

There is no experience in pregnant women who have received this drug.

Precautions. Minor transient elevations of SGOT have occurred in a small percentage of patients. Therefore, this drug should be used with caution in patients with pre-existing liver dysfunction.

Adverse Reactions. The most frequently encountered adverse reactions are related to the gastrointestinal system.

Gastrointestinal and hepatic reactions: anorexia, nausea, vomiting, gastralgia, abdominal cramps, diarrhea and tenesmus, transient elevation of SGOT.

CNS reactions: headache, dizziness, drowsiness, and insomnia.

Skin reactions: rashes.

Dosage and Administration. *Children and Adults:* Antiminth Oral Suspension (50 mg. of pyrantel base/ml.) should be administered in a single dose of 11 mg. of pyrantel base per kg of body weight (or 5 mg /lb.); maximum total dose 1 gram. This corresponds to a simplified dosage regimen of 1 cc. of Antiminth per 10 lbs. of body weight. (One teaspoonful = 5 cc)

Antiminth (pyrantel pamoate) Oral Suspension may be administered without regard to ingestion of food or time of day; and purging is not necessary prior to, during, or after therapy. It may be taken with milk or fruit juices. Because of limited data on repeated doses, no recommendations can be made

How Supplied. Antiminth is available as a pleasant tasting caramel-flavored suspension which contains the equivalent of 50 mg pyrantel base per ml., supplied in 60 cc. bottles.

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(pyrantel pamoate)

equivalent to 50 mg pyrantel/ml

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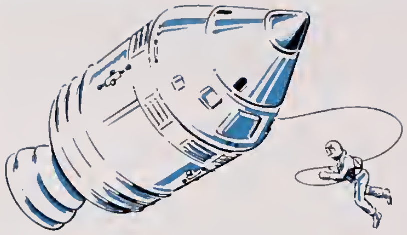

Man in space, now fait accompli, re-emphasizes the importance of Uro-Phosphate therapy. Research into the effect of space travel on the astronaut reveals that weightlessness causes loss of bone calcium. As the bones are required to bear less and less of the weight of the body they lose calcium, increasing the calcium content of the urine. When physical activity is reduced, the acidity of the urine should be adjusted to keep increased calcium in solution . . . . a prophylaxis to prevent kidney or bladder calculi.

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Each tablet contains: METHENAMINE, *300 mg.;* SODIUM ACID PHOSPHATE, *500 mg.*

Uro-Phosphate gives comfort and protection when inactivity causes discomfort in the urinary function. It keeps calcium in solution, preventing calculi; it maintains clear, acid, sterile urine; it encourages complete voiding and lessens frequency when residual urine is present.

Dosage:

For protection of the inactive patient 1 or 2 tablets every 4 to 6 hours is usually sufficient to keep the urine clear, acid and sterile.

2 tablets on retiring will keep residual urine acid and sterile, contributing to comfort and rest.

A clinical supply will be sent to physicians and hospitals on request.

Uro-Phosphate contains sodium acid phosphate, a natural urinary acidifier. This component is fortified with methenamine which is inert until it reaches the acid urinary bladder. In this environment it releases a mild antiseptic keeping the urine sterile.

Uro-Phosphate is safe for continuous use. There are no contra-indications other than acidosis. It can be given in sufficient amount to keep the urine clear, acid and sterile. A heavy sugar coating protects its potency.

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John Parks, M.D.
1908—1972

A familiar figure in the medical community for more than three decades, Dr. John Parks died July 5 after a heart attack. He had served with distinction the causes of sound practice, teaching excellence and the personal nature of medical care. He was a forceful, positive man who brushed aside negative thoughts as irrelevant time wasters and moved to bring consensus among those he led in professional organizations.

Dr. Parks well understood the problems of health care and teaching the ones who deliver it, having advanced through the academic ranks at George Washington University from Adjunct Clinical Professor to Vice President for Medical Affairs. A measure of his flexibility can be seen in the GW Hospital nurseries: he helped design the rooming-in facilities for newborns and their mothers for the new hospital in 1948, and oversaw the plans to remove them in 1972 when trends in infant care rendered them obsolete.

Dr. Parks wrote on privileged communication and counseled medical students on the importance of the "silent language" spoken between a reassuring physician and his worried patients. He assured a maturing population of women that they are "healthier and younger at 50 than ever before in the life of our country," and that it is the responsibility of physicians to help them stay that way. He wrote that a "hospital must be prepared not only to provide the safeguards of a growing science but some of those humanities which distinguish a home from a hotel."

As Dean, Dr. Parks strongly reiterated that the medical schools in the District of Columbia form a vital asset in the city's health resources, and worked in harmony with his fellow Deans at Howard and Georgetown to marshal the educational and training missions of the Universities to help meet urgent and continuing community needs.

John Parks, M.D.

John Parks headed many national organizations in education and medicine and exerted strong influence in the creation of new schools that would "help to provide more physicians for the evergrowing health needs of this country." He believed that federal support of medical education is essential. As President of the Association of American Medical Colleges, he launched with American Medical Association support their historic joint effort in medical education.

On June 30, he delivered the commencement address for house staff at the Bethesda Naval Hospital, in which he contrasted past methods, present trends and 20th Century techniques available to the physician. The teamwork and technology are essential, he said, but "In the solitude of his own mind and thought (the physician) must decide when, what and how to help his patients. He alone is responsible for his words and actions. Only he can do his best and at least do no harm." The speaker had come from a week's hospital confinement to deliver on a commitment.

Dean Parks worked to the end of the plans for the new medical school that will open this year near the University Hospital. "This will be the cover wrapped around the GW curriculum," he was fond of saying. It is the dream coming true for alumni, students and faculty that he toured many times in "hardhat visits." The new school is the synthesis of his legacy as teacher, persuader and administrator.

William Woodrow Stanbro, M.D.
1913–1972

Dr. William W. Stanbro, Professor and Chairman of the Department of Radiology at the George Washington University School of Medicine from 1947 to 1971 died June 23, 1972 in Salina, Kansas.

Born in Ellicottville, New York, he received his M.D. degree from the Washington University School of Medicine in St. Louis. He interned at the St. Louis City Hospital and served residencies at the Robert Koch Hospital in Koch, Missouri and at the Barnes Hospital in St. Louis.

A Captain in the Marine Corps during World War II, Dr. Stanbro served in the European theatre as a radiologist at the 21st General Hospital.

While at George Washington University, he also served as program director of the radiology residency training program and was a consultant in radiology to the National Institutes of Health, D.C. General Hospital and the Veterans Administration. He was senior consultant to the Nuclear Institute in Cleveland and a member of the advisory committee in radiology at the Washington Technical Institute.

He moved to Salina in 1971, where he was a radiologist with the Mowry Clinic.

In addition to his membership in the Medical Society of the District of Columbia, Dr. Stanbro was a member of the American Medical Association, the American College of Radiology, the Society of Nuclear Medicine, the New York Academy of Science, the Society of Chairmen of Academic Radiology Departments and the Association of University Professors. He was a fellow emeritus of the American College of Chest Physicians.

He is survived by his wife, Rosemary, of the home; a son, Dr. William David Stanbro, of Arlington, Virginia; three daughters, Mary Anne Stanbro, of Miami, Virginia Proctor of Falls Church, Virginia, and Carol Jean Stanbro, of Salina; a brother, Forrest Stanbro, of Salamanca, New York, and two grandchildren.

Louis Bernard Bachrach, M.D.
1905–1972

Dr. Louis B. Bachrach, Chief of Urology at Prince Georges General Hospital from its opening until his retirement last year, died June 21, 1972. He resided at 4920 30th Street, N.W. in Washington, D.C.

A native of Washington, he received his undergraduate and M.D. degrees from George Washington University here. He served both his internship and residency at Sinai Hospital in Baltimore.

Dr. Bachrach held the rank of Captain in the medical branch of the Army Air Corps from 1942 to 1946, and established his practice here following his discharge from the military. He maintained a private practice at 919 19th Street, N.W.

An active member of the Medical Society of the District of Columbia, he was an affiliate member of the Prince Georges County Medical Society. He also held membership in the American Medical Association, the Southern Medical Association, the American Board of Urology and the American Urological Association.

Dr. Bachrach is survived by his wife, Ruth, of the home; two children, Marcella Levy, of Chevy Chase, Maryland, and Alan Bachrach, of Newark, Delaware; a brother, Israel Bachrach, of Washington; a sister, Bessie Cohen, of New York, New York, and one grandchild.

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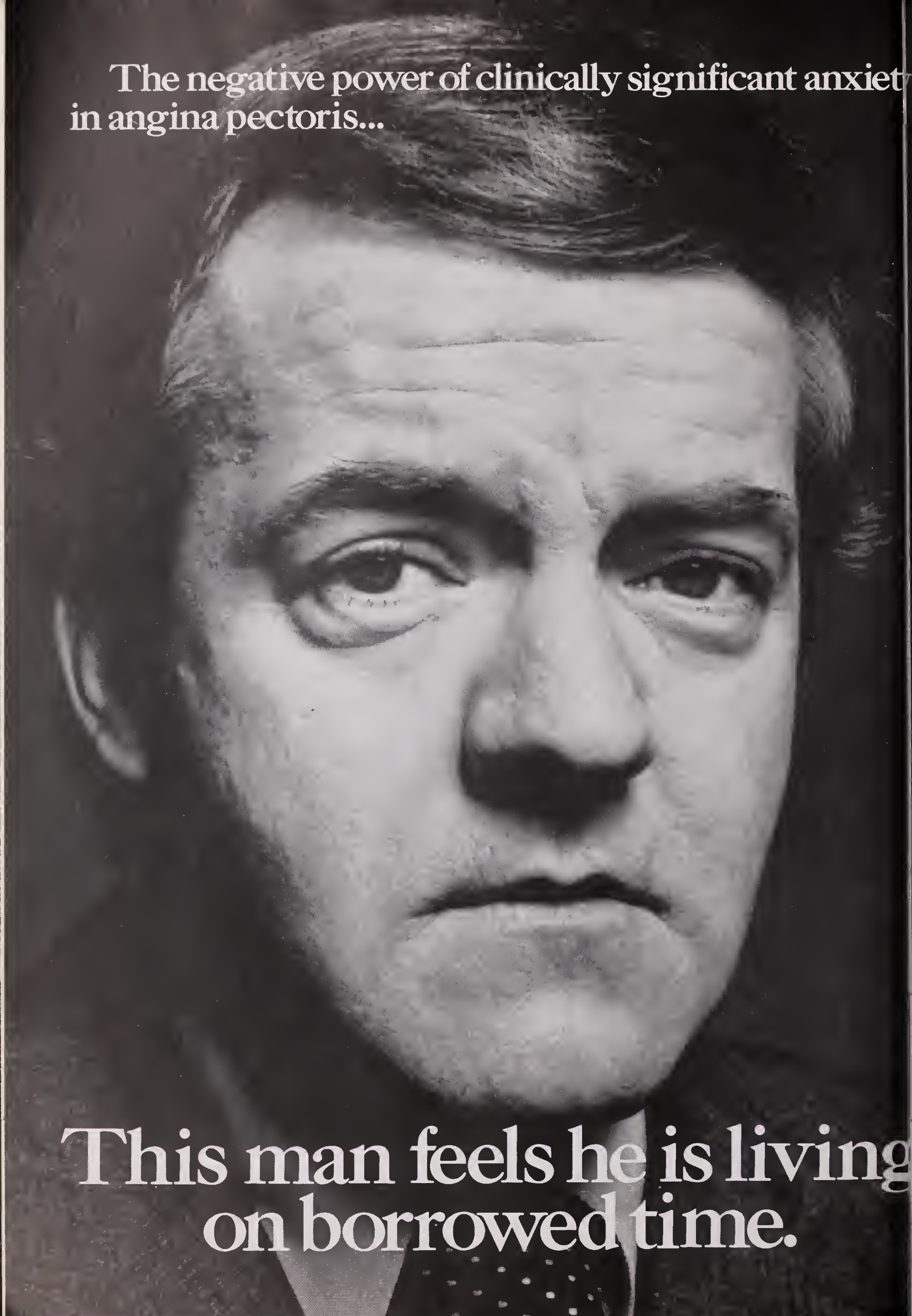
The negative power of clinically significant anxiet
in angina pectoris...
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on borrowed time.

During anginal attacks, patients may suffer intense apprehension. More frequently, however, they experience a continuing sense of less severe but nonetheless disproportionate anxiety.

Reduction of such clinically significant anxiety is important, since undue emotional stress may precipitate further anginal episodes.

Adjunctive Librium (chlordiazepoxide HCl) may be especially suitable for relief of clinically significant anxiety and emotional tension in anginal patients because of its generally prompt therapeutic effectiveness and wide margin of safety. In a recent double-blind randomized study,* Librium (chlordiazepoxide HCl) was administered for relief of moderate anxiety in 20 anginal patients seen in office practice over a 20-week period. Symptoms of emotional distress related to anxiety were rated at base-line, one week, two weeks and monthly thereafter. Relief was obtained notably early in therapy. The clinical results demonstrated that Librium offers the coronary patient an antianxiety drug that, in the author's opinion, is both effective and safe. In general use, the most common side effects reported have been drowsiness, ataxia and confusion, particularly in the elderly and debilitated. (See summary of prescribing information.)

Librium (chlordiazepoxide HCl) is used concomitantly with certain specific medications of other classes of drugs, such as cardiac glycosides, diuretics and antihypertensive agents, whenever anxiety is clinically significant. The drug should be discontinued after anxiety has been reduced to appropriate levels.

The positive power of adjunctive Librium® (chlordiazepoxide HCl)

10-mg, 25-mg capsules
up to 100 mg daily

for moderate to severe anxiety accompanying angina pectoris

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of anxiety and tension occurring alone or accompanying various disease states.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported. Use of any drug in pregnancy, lactation, or in women of childbearing age requires that its potential benefits be weighed against its possible hazards.

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Supplied: Librium® Capsules containing 5 mg, 10 mg or 25 mg chlordiazepoxide HCl. Libritabs® Tablets containing 5 mg, 10 mg or 25 mg chlordiazepoxide.

*Levine, S.: "Angina Pectoris and Emotional Overlay," Scientific Exhibit presented at the Annual Meeting of the Maine Medical Association, Kennebunkport, Me., June 13-15, 1971.

A copy of the Levine study may be obtained from your Roche representative.

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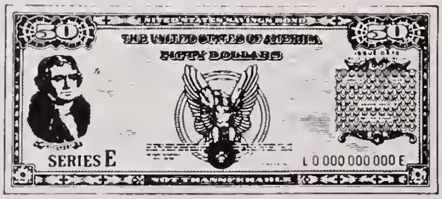

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General Chairman, 1972 Scientific Assembly

Plenary Sessions

Friday, October 27, 1972
9:00 a.m.-10:00 a.m.
Poiitical Candiates—Implications for Physicians
Mr. Harry Hinton
Deputy Director
Public Affairs Division
American Medical Association

Learn the Political Platform of the Candidates You Will be Voting For in November

Saturday, October 28, 1972
9:00 a.m.-10:00 a.m.
Who's Doing What to Whom?
Cortez F. Enloe, Jr., M.D.
Editor: *Nutrition Today*
Enlow, Stalvey & Associates, Inc.
Washington, D.C.

Be Up-To-Date on Diet Fashion!

Sunday, October 29, 1972
9:00 a.m.-10:00 a.m.
Charles A. Hoffman, M.D.
President, American Medical Association

General Sessions

I

Current Views in the Diagnosis, Functional Assessment and Management of Coronary Artery Disease

Friday, October 27, 1972
10:10-12:00 Noon
Chairman: **James M. Bacos, M.D.**
Introduction-**Juan B. Calatayud, M.D.**

Current Views in the Diagnosis of Coronary Heart Disease
David H. Spodick, M.D.

The Value of the Mean QRS and P Axes in the Detection of Coronary Heart Disease
Juan B. Calatayud, M.D.

The Value of the Exercise ECG in the Diagnosis of Coronary Heart Disease
Tazewell Banks, M.D.
Two Case Presentations-Dr. Calatayud
Question and Answer Period

Saturday, October 28, 1972
10:10-12:00 Noon
Chairman: **Jack P. Segal, M.D.**

Noninvasive Functional Assessment in Ischemic Heart Disease
David H. Spodick, M.D.

The Clinical Significance of Mitral Insufficiency in Acute Coronary Heart Disease
James A. Ronan, M.D.

Value and Limitations of Resting 12 Lead ECG in the Diagnosis of Coronary Artery Disease—An ECG-Angiographic Correlation
Jorge C. Rios, M.D.
Two Case Presentations—Dr. Rios
Question and Answer Period

(continued)

Sunday, October 29, 1972
10:10–12:00 Noon

Chairman: **Gerald I. Shugoll, M.D.**

Current Views in the Medical and Surgical Management of Coronary Heart Disease
David H. Spodick, M.D.

Acute Coronary Care—1972
Joseph Lindsay, Jr., M.D.

Current Views in the use of Pacing in Acute Coronary Care
Ross D. Fletcher, M.D.
Two Case Presentations—Dr. Lindsay
Question and Answer Period

Actual cases will be presented by the moderator to the participating panel, with particular attention to Dr. David H. Spodick and the chairman for the day who will be subjected to questioning as to the need and interpretation of data and management of the patient.

II

Heroin-Marijuana-LSD-Alcohol

Friday, October 27, 1972
10:10–12:00 Noon

Moderator: **Marvin C. Korengold, M.D.**

Mind Altering Drugs: Physical Necessity or Emotional Crutch?
Solomon H. Snyder, M.D.
Professor of Pharmacology, Johns Hopkins University School of Medicine

Doing Drugs: A Family Affair
Howard Hoffman, M.D.
Chief Physician, Psychiatric Institute Drug Abuse Center
Question and Answer Period

Saturday, October 28, 1972
10:10–12:00 Noon
Moderator: **Allen M. Mondzac, M.D.**

The Heroin Epidemic
Robert DuPont, M.D.
Director, Narcotic Treatment Administration D.C. Government

Psychedelic Drugs: It's Great to be Alive!
Robert Cope, M.D.
Associate Physician
Narcotic Treatment Administration

Marijuana: Why Isn't it Legal?
Solomon Snyder, M.D.
Question and Answer Period

Sunday, October 29, 1972
10:10–12:00 Noon
Moderator: **Earl H. Mitchell, M.D.**

Man on the Rocks
Paul L. Travis
Director, Rehabilitation Center for Alcoholics—D.C. Government

Can He Get Off?
Maxwell Weisman, M.D.
Director, Maryland Division of Alcoholism Control
Question and Answer Period

III

Human Environment and Pollution

Friday, October 27, 1972
10:10–12:00 Noon
Moderator: **Murdock Head, M.D.**

Overall Problem—Ecology and the Environment
Amos N. Johnson, M.D.
President, American Board of Family Practice
Mr. Lawrence Laurent
T.V. Editor for The Washington Post
Mr. Thomas F. Williams
Acting Director, Office of
Solid Waste Managment Programs
Environmental Protection Agency
Question and Answer Period

Saturday, October 28, 1972
10:10–12:00 Noon
Moderator: **Mr. Malcolm Hope**

Fact or Fancy in Consumer Products
What We Really Need to Know—Fact from Fancy
Albert C. Kolbye, M.D.
Deputy Director
Food and Drug Administration

Prophylactic and Regulatory Steps to be Considered
Mr. Thomas Williams
Resource Person
D.C. Department of Economics
Question and Answer Period

Sunday, October 29, 1972
10:10–12:00 Noon
Moderator: **Clifton Gruver, M.D.**

Solid Waste—Will it Consume Us?
The Problem on a National Basis
Mr. Thomas F. Williams
The Problem on a Local Basis
Mr. Malcolm Hope
Director, Environmental Planning
D.C. Department of Human Resources
Public Acceptance of the Solution to Solid Waste Management Problem
Mr. William M. Harrington, Jr.
Consulting Engineer
Whitman, Requardt & Associates
Question and Answer Period

IV

New Drugs, Drug Interaction and Medical Controversies

Friday, October 27, 1972
10:10–12:00 Noon
Moderator: **Jay R. Shapiro, M.D.**

Current Topics in Antibiotic Use
Christopher M. Martin, M.D.
Senior Director of Medical Affairs
Merck, Sharp and Dohme

Antibiotic Considerations in Patients with a Prosthetic Valve
Herbert L. Tannenbaum, M.D.
Clinical Assistant Professor of Medicine
Georgetown University School of Medicine
Question and Answer Period

Saturday, October 28, 1972
10:10–12:00 Noon
Moderator: **Jay R. Shapiro, M.D.**

Clinical Problems of Drugs Interaction
Elliot S. Vesell, M.D.
Professor and Chairman
Department of Pharmacology
University of Pennsylvania at Hershey
Question and Answer Period with
Christopher M. Martin, M.D.

Sunday, October 29, 1972
10:10–12:00 Noon
Moderator: **Jay R. Shapiro, M.D.**

Drug Interaction in Endocrinology and Diabetes
Norman H. Ertel, M.D.
Professor of Medicine, New Jersey
College of Medicine and Dentistry

Therapeutic Controversies in Thyroid Disease
Jay R. Shapiro, M.D.
Assistant Professor of Medicine
George Washington University School of Medicine
Chairman, Department of Endocrinology
Washington Hospital Center
Question and Answer Period

V

Sex, Venereal Disease and Abortion

Friday, October 27, 1972
10:10–12:00 Noon

Moderator: **Julius Fogel, M.D.**

Sexual Adequacy and Inadequacy
Masters & Johnson Technique
Sallie S. Schumacher, Ph.D.
Director, Program in Human Sexuality
Long Island Jewish Medical Center
Julius Fogel, M.D.
Assistant Clinical Professor
of Obstetrics and Gynecology

George Washington University School of Medicine
Reverened Knox Kreutzer
President, Marriage & Family Institute, Inc.
Washington, D.C.
Question and Answer Period

Saturday, October 28, 1972
10:10–12:00 Noon
Moderator: **Julius Fogel, M.D.**

Medical Aspects— Venereal Disease
Julius Fogel, M.D.
Reverend Knox Kreutzer
Sallie S. Schumacher, Ph.D.
Question and Answer Period

Sunday, October 28, 1972
10:10–12:00 Noon
Moderator: **Julius Fogel, M.D.**

Historical, Sociological and Moral Aspects
Reverend Knox Kreutzer
Sallie S. Schumacher, Ph.D.
Julius Fogel, M.D.
Question and Answer Period

What have Masters & Johnson really contributed in their research in sexual function? What does their therapy accomplish and what techniques and methods are used? What does Masters and Johnson's "Successful Therapy" *not* accomplish between the partners?

VI
Surgical Controversies

Friday, October 27, 1972
10:10–12:00 Noon
Moderator: **LaSalle D. Leffall, Jr., M.D.**

Primary Resection for Perforated Colon Lesions
John L. Madden, M.D.
Director, Department of Surgery
St. Clare's Hospital
PANEL DISCUSSION
Phillip H. Philbin, M.D.
Senior Attending Surgeon
Washington Hospital Center
William S. McCune, M.D.
Clinical Professor of Surgery
George Washington University School of Medicine
Bernard Peacock, M.D.
Chief, Department of Surgery
Morris Cafritz Memorial Hospital

Saturday, October 28, 1972
10:10–12:00 Noon
Moderator: **John Potter, M.D.**

Electro-Coagulation in the Treatment of Cancer of the Rectum
John L. Madden, M.D.
PANEL DISCUSSION
Alejandro F. Castro, M.D.
Senior Attending Surgeon
Suburban Hospital
Karl C. Jonas, M.D.
Chief, Department of Surgery
Doctors Hospital
Jack E. White, M.D.
Professor of Surgery
Cancer Coordinator
Howard University College of Medicine

Sunday, October 29, 1972
10:10–12:00 Noon
Moderator: **William W. Funderburk, M.D.**

Modified Radical Mastectomy for Cancer of the Breast
John L. Madden, M.D.
PANEL DISCUSSION
Louis J. Gofreddi, M.D.
Chief, Department of Surgery
Providence Hospital
Ernest A. Gould, M.D.
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Associate Professor of Surgery
George Washington University School of Medicine

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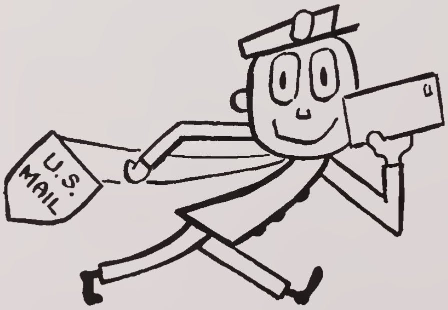

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Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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of the district of columbia

Medico-Legal Report—
"The Physician as
Witness—The Problem
of Dying Declarations"

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PRESIDENT'S MESSAGE

For everything there is a season,
And a time for every matter under heaven;

A time to keep silent, and a time to speak.

Most physicians seem to enjoy the time for speaking more than the time for keeping silent - and at the Annual Business Meeting scheduled for Monday, November 20, members will have an opportunity to speak at some length on topics pertinent to the future of our Society. After the election of officers has been completed an open forum is planned to allow members time for expressing opinions on future Society policies.

The first is the matter of finances. Fortunately, the general budget of the Society has been in the black this year - thanks to the dues increase and the astute management of our Secretary-Treasurer, Executive Director, Franc Ferraraccio. A 5.5 per cent increase to take care of expected and normal increases of operation should keep the 1973 budget balanced. But maintaining the Medical Annals at its current high peak of perfection is a more precarious matter. Unforeseen increases in the cost of paper and printing, coupled with a significant drop in advertising revenues, have resulted in a deficit of major proportions. Only by means of a special assessment can the present format be continued. An alternate solution, such as bi-monthly publication, might be considered. In view of the very favorable response and wide acceptance of the "new" Annals, these alternatives seem unwise - but it is a decision the membership should make.

arrell C. Crain, M.D.

Another major matter of discussion at the Annual Meeting will be the establishment of The Medical Foundation of Metropolitan Washington. As discussed in this page last May, an ad hoc committee has been working for over two years to develop a plan suitable for our members. Full details will be available at the meeting, including why the Foundation is considered necessary, what it is expected to accomplish, and the exact manner in which it will operate. Of interest is the fact that the Prince Georges Medical Society and the Montgomery County Medical Society have already incorporated a Foundation.

Physicians wishing to discuss the new "HMO type" groups of medical practice which are developing in our

Water Treatment and Examination (Holden) Reviewed by Stuart H. Danovitch, M.D.

Living with Your Bad Back (Berland and Addison) Reviewed by Margaret Kenrick, M.D.

A Civilian Doctor in Vietnam (Gloeckner) Reviewed by William W. Funderburk, M.D.

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ANNUAL BUSINESS MEETING

November 20, 1972

The Sheraton-Park Hotel

Reception
6:00 p.m.

Dinner
6:45 p.m.

Meeting
8:00 p.m.

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of the district of columbia

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city will also be given a chance to express their views. Considerable confusion exists as to the relationship of insurance carriers, as well as our Society, to these organizations. In this regard it is pertinent to point out that many of the physicians involved in the formation of these groups (as well as physicians on the staff of Group Health Association) are members in good standing of our Society. As such, their opinions are entitled to the same consideration as those of members practicing in the fee for service tradition.

So the call is sounded for the Annual Meeting and since the Committee on Constitution and Bylaws has no revisions to recommend, the entire meeting will be devoted to discussing vital projects. Come and let the officers know what you are thinking - or forever hereafter hold your peace.

Darrell C. Crain

DEPRESSION AND PUBLIC OFFICE

The recent disclosure of a history of recurrent depression by a political candidate has brought into focus some of the controversial issues that face the field of psychiatry. The question of an individual's ability to exercise responsibility and good judgment while suffering from depression is an aspect of larger issues under discussion. Psychiatrists continue to debate the degree to which individuals have rational, responsible control over their actions, and the degree to which psychological forces within the individual determine what he thinks and what he does. Also debated is whether psychiatric disorders exist at all or whether they are the product of psychiatrists' imaginations and society's convenience. Non-psychiatric physicians asked to comment on such issues are frequently confounded by the contradictory and often unscientific data. How can one approach this morass?

Medicine has a tradition of being both empirical and scientific. As physicians we are presented with clinical problems which we attempt to understand and treat as best we can, by making careful observations and drawing the most rational conclusions possible. We must proceed utilizing the available data and methods, however inadequate, to approach the problem at hand. Our hypotheses, methods, and conclusions are submitted to scientific study in order to test and expand our knowledge. It is by these means that medicine has been able to develop the ability to approach clinical problems with increasing effectiveness.

Psychiatry is no different. It is more complex in that the biological, psychological and social factors involved are more difficult to understand and to integrate into a meaningful scheme. Clinical psychiatry is largely at an empirical descriptive stage. Syndromes are described, the patterns of development of these syndromes are observed, causal factors are hypothesized and what appears to work empirically is used for treatment. The scientific research which attempts to further the understanding of this empirical system develops much interesting data but is still of limited use in the clinical situation. We are, then, left with needing to be definite where there is a lack of clarity, to draw conclusion from incomplete data, and to commit ourselves to positions with the realization that future data might show our conclusions to be in error.

What can we say about the effect of psychiatric disorders in general and recurrent depression, specifically on the capacity of the individual to exercise responsibility and good judgment? Our observations tell us that some types of psychiatric disorders produce major changes in the person's observable behavior, his emotional state, or his thought processes. The latter are characterized by less drastic changes. Individuals are rendered uncomfortable by symptoms, often depressive, or by maladaptive patterns of behavior, but are usually able to function in a responsible, consistent way during the course of such disorders. These are often more like "problems in living" than true "diseases."

We do know that depressive disorders that are recurrent and require hospitalization and/or electroconvulsive therapy are more likely to be of a former variety, that is, productive of major changes of the individual's psychic equilibrium while he is ill. Moreover, we know that recurrent depressions have a tendency to continue to recur despite treatment methods available today. We do not know the details of the disorder suffered by Senator Eagleton. Based on what we do know, his history of recurrent depression certainly does not disqualify him from holding positions of responsibility. The position of Vice-President of the United States is one of such extreme responsibility, however, that it should be held by a person in whom there is minimal risk of an occurrence of major psychiatric disorder.

David H. Fram, M.D.
Director, Psychiatric Institute
Drug Abuse Rehabilitation Center
Assistant Clinical Professor
of Psychiatry
George Washington University
School of Medicine

HELP DETERMINE YOUR FUTURE

It is heard or read, all too commonly, that our health care system is a "nonsystem" not geared to the real needs of all people. And there are other attacks which alledgedly degrade the quality and character of the practice of medicine today. This so-called "nonsystem" of medical care is based on the American system of free enterprise, pluralism, fee-for-service, etc., and needs to be defended with truth and vigor.

Generally, physicians claim they are too busy with professional matters to become involved in the political arena where crucial decisions concerning the practice of medicine and the delivery of medical care are now under discussion. While the physician is admittedly busy with his practice of medicine, one needs to consider the astounding fact that these discussions are being held by a great number of people who know very little about the practice of medicine. These are the people who, if not advised, will determine how the future practice of medicine shall be conducted. It is clearly evident that unless physicians emerge collectively from their professional isolationism, the result may be distinctly adverse, not only from the standpoint of the profession, but more importantly from the standpoint of the public.

A major influence in contemporary American life which has relevance to this situation is the uneasy relationship between the individual and society's institutions, particularly the State. For the past fifty years there has been a continuing upward flow of power to large institutional units, whether they be corporate conglomerates, unions, professional associations, or the government. There has been created during the course of these fifty years, a culture which requires the individual to submit to society in ways that formerly he did not have to do. Large institutions require multiple layers of procedures and rules which restrict individual freedom. Intuitively, therefore, the individualist feels that bureaucracies must make man into an object in order to deal with him, so that there is a depersonalization at the time when the individualist is casting about for identity.

Recent studies have shown that the physician enjoys the highest profile of all the various professional images. In the public eye he stands first in trust, confidence and service. This should not be surprising to anyone. (On the other hand, it would be extremely surprising if it were the reverse.) It is also not surprising that the President has referred to the physician and more directly to the AMA as "one great source of towering strength."

Each physician should accept the cloak of his "image" both as an individual and as a member of his "professional institution" and emerge from his professional isolationism. As pointed out earlier, our culture today is rooted to the actions of mass "institutions." Our political leaders - those who are discussing the future of medicine - are extremely sensitive to organized opinion, especially when it represents a broad spectrum of constituents. The best way to influence the political leader is to influence his constituent. The physician's strength, both as an individual and as a member of organized medicine is conducive to the exercise of this influence.

F.P. Ferraraccio
Executive Director

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Precautions: Do periodic serum electrolyte and BUN determinations. Do periodic hematologic studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in postsympathectomy patients. The following may occur: hyperuricemia and gout, reversible nitrogen retention, decreasing alkali reserve with possible metabolic acidosis, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), digitalis intoxication (in hypokalemia). Use cautiously in surgical patients. Concomitant use with antihypertensive agents may result in an additive hypotensive effect.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis; rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting (may indicate electrolyte imbalance), diarrhea, constipation, other gastrointestinal disturbances. Rarely, necrotizing vasculitis, paresthesias, icterus, pancreatitis, and xanthopsia have occurred with thiazides alone.

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IN EDEMA*—IN HYPERTENSION*

If you've seen one, have you really seen them all?

The following patient profiles represent typical clinical situations, but do not necessarily represent actual cases.

Age 22, previously normal mens with occasional menorrhagia. No on a sequential O.C. for four mor Complains of heavy flow, occasional intracyclic bleeding, edema, tender swollen breasts.

Indicates estrogen excess.

1st choice: Switch to a combination 50-mcg.-estrogen O.C. (such as **Demulen***).

Age 19, small breasts, minor hirsutism, oily hair and skin. History of metrorrhagia, skipped or scanty menses. New user.

Indicates androgenic excess or estrogen deficiency (fertility is suspect).

1st choice: An estrogen-dominant O.C. (such as **Enovid-E***).

Age 25, average frame, poor complexion. No problem with menses, normal para 1. On a low-estrogen/high-progestogen O.C. for two years. Now complains of scanty flow, decreased libido, depression.

Indicates probable buildup of progestogen-related side effects.

1st choice: Switch to a center-spectrum O.C. with more estrogen, less progestational activity (such as **Ovulen***).

Age 21, short, mammose, with normal menses, some acne. Was put on prenuptial regimen of 50-mcg.-estrogen/moderate-progestogen O.C. for two months. Now has increased acne.

Indicates metabolic production of androgen or relative estrogen deficiency.

1st choice: Switch to a 100-mcg.-estrogen combination (such as **Enovid-E*** or a sequential).

Unmasked, physiologically and anatomically, they're not all the same. A basic difference lies in their hormone profiles. One may secrete too much estrogen, another not enough...or perhaps too much androgen; the vast majority would fit somewhere into the broad center spectrum.

Although the profiles described below may not be completely predictive, in optimal O.C. selection, the estrogen-progestogen activity ratio should be carefully matched to the patient profile. Searle offers you O.C.s in a range not only suitable for your patients in the balanced center spectrum, but also adaptable to the patient with another type of hormone profile.

Oral contraceptives are complex medications. Among the commonly reported adverse reactions are: intracycle bleeding, fluid retention, tender or swollen breasts, exacerbation of acne condition, changes in libido, amenorrhea while on medication and upon discontinuance, nausea, leg cramps, headaches, weight gain. Therefore, after reference to the prescribing information, oral contraceptives should be prescribed with care.

*Note: In some patients any level of exogenous estrogen or progestogen may produce symptoms of excess hormone activity.

e 25, tall, slender, athletic,
th flat chest. On a progestogen-
minant 50-mcg.-estrogen O.C.
s recurrent trichomoniasis
d Monilia.
Indicates estrogen deficiency and
cess of progestogen in current O.C.
1st choice: Switch to a com-
ation pill with 100 mcg.
trogen and less progestational
tivity (such as **Enovid-E**® or
ulen® or a sequential).

Age 23, "Miss America" figure, previously normal menses, healthy skin and hair. On a 50-mcg.-estrogen pill for four months. Complains of intracyclic bleeding.

Indicates probable need for more estrogen.

1st choice: Switch to a center-spectrum O.C. with more estrogen and moderate progestogen dominance (such as **Ovulen**®).

Age 21, college senior, average build. On highly progestogen-dominant/low-dose-estrogen O.C. for six months. Now complains of amenorrhea, between-cycle headaches, weight gain.

Indicates probable progestogen excess.

1st choice: Switch to a center-spectrum pill (such as **Ovulen**®).

Age 27, slightly overweight, multiparous. Nausea with all three pregnancies and with a sequential O.C. three years ago. Has premenstrual fluid retention and leg cramps.

Indicates probable excess of estrogen

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Both Ovulen and Demulen are available in 21- and 28-pill schedules.

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a brief summary
rescribing information,
ase see next page.

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Actions–Ovulen and Demulen act to prevent ovulation by inhibiting the output of gonadotropins from the pituitary gland. Ovulen and Demulen depress the output of both the follicle-stimulating hormone (FSH) and the luteinizing hormone (LH).

Special note–Oral contraceptives have been marketed in the United States since 1960. Reported pregnancy rates vary from product to product. The effectiveness of the sequential products appears to be somewhat lower than that of the combination products. Both types provide almost completely effective contraception.

An increased risk of thromboembolic disease associated with the use of hormonal contraceptives has now been shown in studies conducted in both Great Britain and the United States. Other risks, such as those of elevated blood pressure, liver disease and reduced tolerance to carbohydrates, have not been quantitated with precision.

Long-term administration of both natural and synthetic estrogens in subprimate animal species in multiples of the human dose increases the frequency of some animal carcinomas. These data cannot be transposed directly to man. The possible carcinogenicity due to the estrogens can be neither affirmed nor refuted at this time. Close clinical surveillance of all women taking oral contraceptives must be continued.

Indication–Ovulen and Demulen are indicated for oral contraception.

Contraindications–Patients with thrombophlebitis, thromboembolic disorders, cerebral apoplexy or a past history of these conditions, markedly impaired liver function, known or suspected carcinoma of the breast, known or suspected estrogen-dependent neoplasia and undiagnosed abnormal genital bleeding.

Warnings–The physician should be alert to the earliest manifestations of thrombotic disorders (thrombophlebitis, cerebrovascular disorders, pulmonary embolism and retinal thrombosis). Should any of these occur or be suspected the drug should be discontinued immediately.

Retrospective studies of morbidity and mortality conducted in Great Britain and studies of morbidity in the United States have shown a statistically significant association between thrombophlebitis, pulmonary embolism, and cerebral thrombosis and embolism and the use of oral contraceptives. There have been three principal studies in Britain[1-3] leading to this conclusion, and one[4] in this country. The estimate of the relative risk of thromboembolism in the study by Vessey and Doll[3] was about sevenfold, while Sartwell and associates[4] in the United States found a relative risk of 4.4, meaning that the users are several times as likely to undergo thromboembolic disease without evident cause as nonusers. The American study also indicated that the risk did not persist after discontinuation of administration and that it was not enhanced by long-continued administration. The American study was not designed to evaluate a difference between products. However, the study suggested that there might be an increased risk of thromboembolic disease in users of sequential products. This risk cannot be quantitated, and further studies to confirm this finding are desirable.

Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions medication should be withdrawn.

Since the safety of Ovulen and Demulen in pregnancy has not been demonstrated, it is recommended that for any patient who has missed two consecutive periods pregnancy should be ruled out before continuing the contraceptive regimen. If the patient has not adhered to the prescribed schedule the possibility of pregnancy should be considered at the time of the first missed period.

A small fraction of the hormonal agents in oral contraceptives has been identified in the milk of mothers receiving these drugs. The long-range effect to the nursing infant cannot be determined at this time.

Precautions–The pretreatment and periodic physical examinations should include special reference to the breasts and pelvic organs, including a Papanicolaou smear since estrogens have been known to produce tumors, some of them malignant, in five species of subprimate animals. Endocrine and possibly liver function tests may be affected by treatment with Ovulen or Demulen. Therefore, if such tests are abnormal in a patient taking Ovulen or Demulen, it is recommended that they be repeated after the drug has been withdrawn for two months. Under the influence of progestogen-estrogen preparations preexisting uterine fibromyomas may increase in size. Because these agents may cause some degree of fluid retention, conditions which might be influenced by this factor, such as epilepsy, migraine, asthma, cardiac or renal dysfunction, require careful observation. In breakthrough bleeding, and in all cases of irregular bleeding per vaginam, nonfunctional causes should be borne in mind. In undiagnosed bleeding per vaginam adequate diagnostic measures are indicated. Patients with a history of psychic depression should be carefully observed and the drug discontinued if the depression recurs to a serious degree. Any possible influence of prolonged Ovulen or Demulen therapy on pituitary, ovarian, adrenal, hepatic or uterine function awaits further study. A decrease in glucose tolerance has been observed in a significant percentage of patients on oral contraceptives. The mechanism of this decrease is obscure. For this reason, diabetic patients should be carefully observed while receiving Ovulen or Demulen therapy. The age of the patient constitutes no absolute limiting factor, although treatment with Ovulen or Demulen may mask the onset of the climacteric. The pathologist should be advised of Ovulen or Demulen therapy when relevant specimens are submitted. Susceptible women may experience an increase in blood pressure following administration of contraceptive steroids.

Adverse reactions observed in patients receiving oral contraceptives–A statistically significant association has been demonstrated between use of oral contraceptives and the following serious adverse reactions: thrombophlebitis, pulmonary embolism and cerebral thrombosis.

Although available evidence is suggestive of an association, such a relationship has been neither confirmed nor refuted for the following serious adverse reactions: neuro-ocular lesions, e.g., retinal thrombosis and optic neuritis.

The following adverse reactions are known to occur in patients receiving oral contraceptives: nausea, vomiting, gastrointestinal symptoms (such as abdominal cramps and bloating), breakthrough bleeding, spotting, change in menstrual flow, amenorrhea during and after treatment, edema, chloasma or melasma, breast changes (tenderness, enlargement and secretion), change in weight (increase or decrease), changes in cervical erosion and cervical secretions, suppression of lactation when given immediately post partum, cholestatic jaundice, migraine, rash (allergic), rise in blood pressure in susceptible individuals and mental depression.

Although the following adverse reactions have been reported in users of oral contraceptives, an association has been neither confirmed nor refuted: anovulation post treatment, premenstrual-like syndrome, changes in libido, changes in appetite, cystitis-like syndrome, headache, nervousness, dizziness, fatigue, backache, hirsutism, loss of scalp hair, erythema multiforme, erythema nodosum, hemorrhagic eruption and itching.

The following laboratory results may be altered by the use of oral contraceptives: hepatic function: increased sulfobromophthalein retention and other tests; coagulation tests: increase in prothrombin, Factors VII, VIII, IX and X; thyroid function: increase in PBI and butanol extractable protein bound iodine, and decrease in T^3 uptake values; metyrapone test and pregnanediol determination.

References: 1. Royal College of General Practitioners: Oral Contraception and Thrombo-Embolic Disease, J. Coll. Gen. Pract. *13*:267-279 (May) 1967. **2.** Inman, W. H. W., and Vessey, M. P.: Investigation of Deaths from Pulmonary, Coronary, and Cerebral Thrombosis and Embolism in Women of Child-Bearing Age, Brit. Med. J. *2*:193-199 (April 27) 1968. **3.** Vessey, M. P., and Doll, R.: Investigation of Relation Between Use of Oral Contraceptives and Thromboembolic Disease. A Further Report, Brit. Med. J. *2*:651-657 (June 14) 1969. **4.** Sartwell, P. E.; Masi, A. T.; Arthes, F. G., Greene, G. R., and Smith, H. E.: Thromboembolism and Oral Contraceptives. An Epidemiologic Case-Control Study, Amer. J. Epidem. *90*:365-380 (Nov.) 1969.

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Actions–Enovid-E acts to prevent ovulation by inhibiting the output of gonadotropins from the pituitary gland. Enovid-E depresses the output of both the follicle-stimulating hormone (FSH) and the luteinizing hormone (LH).

Indication–Enovid-E is indicated for oral contraception.

The *Special Note, Contraindications, Warnings, Precautions* and *Adverse Reactions* listed above for Ovulen and Demulen are applicable to Enovid-E and should be observed when prescribing Enovid-E.

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"The history of science, and in particular the history of medicine . . . is . . . the history of man's reactions to the truth, the history of the gradual revelation of truth, the history of the gradual liberation of our minds from darkness and prejudice."

—George Sarton, from "The History of Medicine Versus the History of Art"

Are combination drug products useful in treatment involving concomitant use of two or more drugs?

Opinion

Results of a questionnaire to 7,000 physicians:

62.9%
Believe combination drug products are useful.

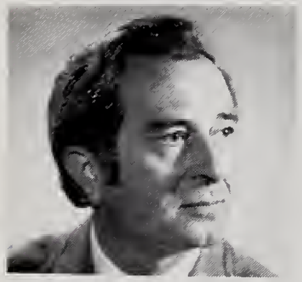
13.8%
Do not believe combination drug products are useful.

Are combination drug product useful in treatment involvin concomitant use of two or more drugs

Opinion & Dialogue Dialogu

Doctor of Medicine

Louis Lasagna, M.D.
Professor and Chairman
Department of
Pharmacology & Toxicology
University of Rochester
School of Medicine
and Dentistry



Obviously, many drugs are given concomitantly. Whether it makes sense to combine medications in one preparation, be it capsule, tablet, or liquid, is a question that can be answered only by examining the advantages and disadvantages in the individual case.

Among the advantages is, first of all, convenience. The more medications that are taken concurrently and the more complicated the directions, the less likely the patient is to take medications accurately. From the standpoint of convenience and accuracy, and economy as well, you can make an important case for putting medications together in one preparation, as long as they are compatible.

By the same token, when you prescribe a properly tested and rational combination, you should have less worry about pharmaceutical or pharmacological compatibility – and about reasonable dosage ratios as well. Compatibility of the formulation should be demonstrated in the laboratory and clinic before the product is available for prescription—which is more than can usually be said for the physician's own spontaneous creations. And, the dosage ratios employed in rational precompounded combinations are designed to meet the needs of substantial numbers of "typical" patients.

There is no doubt that many "atypical" patients are to be found, and for them the prefabricated combination must be rejected. But that hardly argues for eliminating rational combinations from the market. Think, for example, of the problems that would arise if the components of widely accepted combinations, like the oral contraceptives and the diuretic-antihypertensives, always had to be prescribed, purchased and ingested separately.

One disadvantage that comes to mind is some doctors' unawareness of the ingredients a given combination contains. For example, a doctor might know that a patient is allergic to aspirin but forget that a certain analgesic mixture, which he knows only by its trade name, contains aspirin. His prescription, then, causes considerable discomfort, to say the least. This problem is a function of physician education, rather than of combination therapy as such. Improving doctors' knowledge about all medicaments they prescribe is a problem that deserves tackling on its own.

Another accusation leveled at combination drugs is that they encourage sloppiness of diagnosis and treatment. In many cases, however, a combination may prove to be the most effective choice. A good example of the usefulness of combinations appears in a recent article in the *Journal of Chronic Diseases* on the efficacy and side effects of an antihypertensive containing three ingredients, in which the track records of the combination drug and the individual ingredients were compared. Interestingly enough, whether the drugs were given individually or together, incidence and severity of side effects were the same. But blood pressure control was invariably better when the drugs were taken in one combination tablet than when they were taken separately (in "titratable" dosage) or in two or three different tablets.

Deciding which combinations constitute rational therapy obviously leads to a discussion of who is to determine which should be used and which should not. Realistically, I think combinations should be evaluated somewhat differently if they are old and established or new and untried.

In today's regulatory atmosphere, there is no possibility of a new combination being put on the market without a substantial amount of acceptable evidence in the form of controlled trials that show it to be safe and efficacious. On the other hand, I believe a different set of standards should apply to combination preparations that have been around for a long time. In other words, physician acceptance over a long period should be given some weight as evidence of the efficacy and safety of these drugs.

The FDA, however, does not seem to share this attitude. It often requires, for these older products, controlled trials that will monopolize the time of already overtired investigators and cost a greal
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Maker of Medicine

. Clarke Wescoe, M.D.
President
inthrop Laboratories

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cri sm generated against combinations generally. Obviously, no one should be exposed willy-nilly to the potential side effects of two or three antibiotics when only one is needed. At the same time there are cases where it is prudent to prescribe more than one. The clinician is the judge in these circumstances, as he should be.

There is no clear definition of the word rational. Most persons, I suppose, would find it synonymous with reasonable, but in many circumstances it may best be defined as the opinion of those in power at the moment.

Other factors govern combination therapy, not the least of which has been its broad use by practicing physicians anxious to achieve convenience in prescribing, to reduce medication error, and to save money for their patients. Combinations clearly have met the test on all three counts.

I have been impressed by studies showing that the rate of error climbs markedly with the number of medications to be taken, even with sophisticated patients. When medically justified, therefore, this factor alone supports the logic of combination therapy.

The cost argument for combinations appears to be irrefutable. In 1971, R. A. Gosselin studied the 71 combination products (excluding oral contraceptives) among the 200 most prescribed drugs. The study found that if all 71 products were discontinued, and if each ingredient in these combinations were prescribed separately, the price of medicines to patients would jump by $443.2 million on a national basis! At a time when the cost of medical care is under so much fire, it would be nonsensical to boost costs without clearly irrefutable medical reasons.

The part played by government on this question, of course, is fundamental. The FDA should play a role in determining which combinations are reasonable. That role, as defined by law and regulation, is to ensure that any medication on the market is safe and effective in line with its label claims. Certainly combinations are entitled to as much consideration as single entities—neither more nor less. So long as the addition of one drug to another does not make either less safe, or less effective, so long as they are compatible in a formulation, we have a reasonable product. It makes no sense to recommend the use of two products for certain conditions and to deny their being combined in a single form. An unhappy side effect of the problem concerns the efficacy panel discussions of many products submitted for review. The term "effective, but" has been freely interpreted to mean "ineffective" in toto, regardless of the merit of the individual drugs. This interpretation has placed numerous useful combination products in needless jeopardy.

In reading the actual reports of the review panels, it seems clear that some of the ratings were based less on scientific research and clinical observation than on the "informed" opinions of the panelists. These "informed" opinions were accepted at face value, while the "informed" opinions of others who had used the products were rejected. All of this put combination products into a sort of scientific never-never land.

It should be kept in mind by all, government as well as others involved in our health care system, that advances in therapy are seldom made in leaps and bounds but rather by small painstaking steps—and that some of these steps have resulted from research in combination drugs as well as with single entities. Given the near-infinite biologic variation in patient response, this is hardly surprising to clinicians. It should not be to regulatory agencies either.

In the end, the practicing physician is in the best position to decide if a particular combination makes sense. Such a decision should not be made exclusively by those whose responsibility for continuing clinical care is limited. Clinicians are the best judges of efficacy because the ultimate proof of any product's effectiveness is acceptance by physicians who have observed its actions in patients over time. The corollary statement may be made about over-the-counter medicines, which would not long survive if they failed to afford the relief the user anticipates. That the antihistamine in a "cold" remedy may not *always* be necessary is no reason to proscribe the combination generally.

Opinion & Dialogue

What is your opinion, doctor?
We would welcome your comments.

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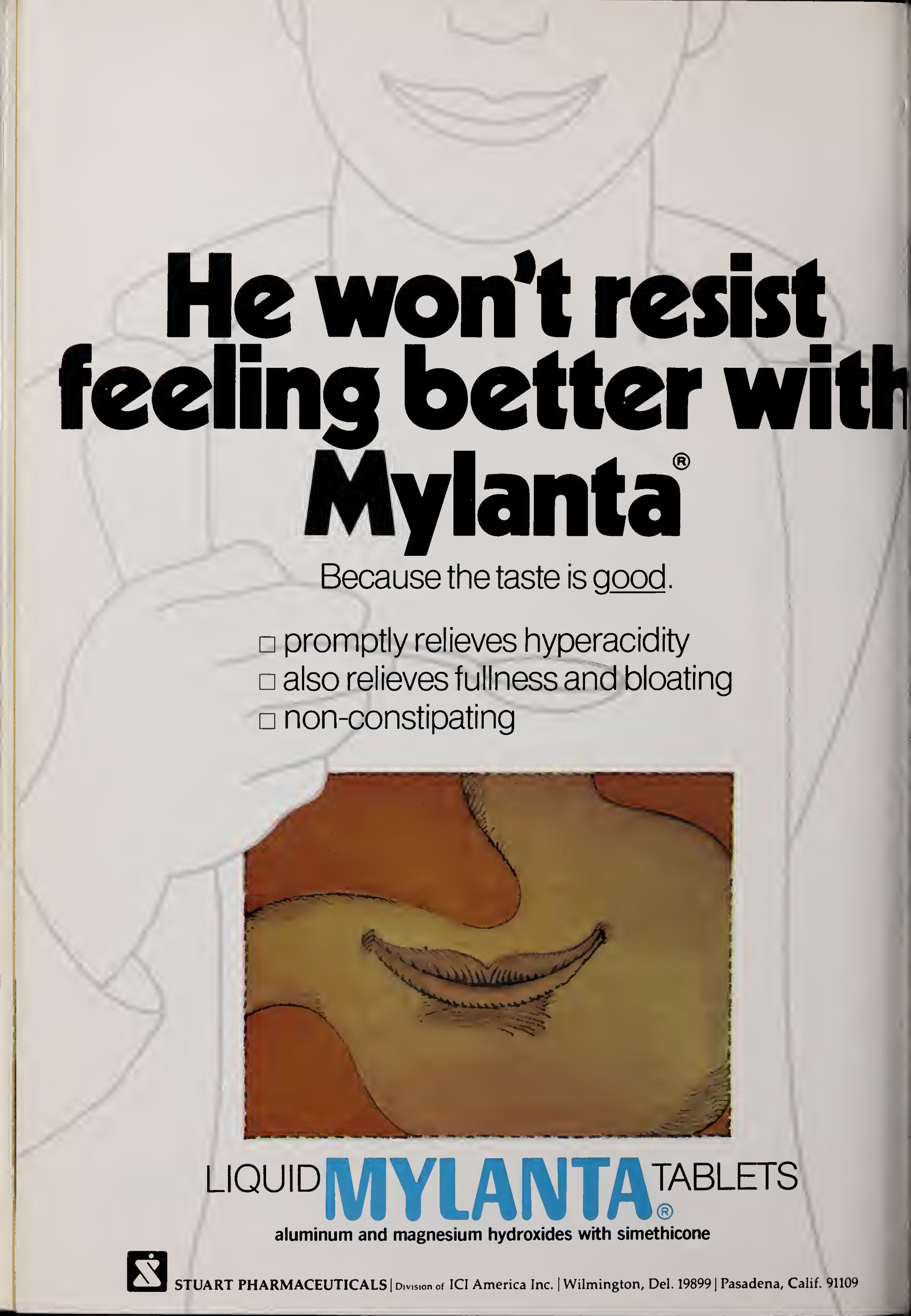

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Highly effective. Active against pinworm...and roundworm.

Non-staining. Doesn't stain teeth or oral mucosa on ingestion. Doesn't stain stools, clothing or linen.

Simple dosage. Single-dose regimen: 1 cc. per 10 lbs. of body weight (1 tsp. per 50 lbs.).

Well-tolerated. Based on pre-introductory studies.

Pleasant-tasting. Easy-to-take, caramel-flavored oral suspension.

Economical. One prescription for the entire family.

Antiminth (pyrantel pamoate) Oral Suspension

Actions. Antiminth (pyrantel pamoate) has demonstrated anthelmintic activity against *Enterobius vermicularis* (pinworm) and *Ascaris lumbricoides* (roundworm). The anthelmintic action is probably due to the neuromuscular blocking property of the drug.

Antiminth is partially absorbed after an oral dose. Plasma levels of unchanged drug are low. Peak levels (0.05-0.13 μg/ml) are reached in 1-3 hours. Quantities greater than 50% of administered drug are excreted in feces as the unchanged form, whereas only 7% or less of the dose is found in urine as the unchanged form of the drug and its metabolites.

Indications. For the treatment of ascariasis (roundworm infection) and enterobiasis (pinworm infection).

Warnings. *Usage in Pregnancy:* Reproduction studies have been performed in animals and there was no evidence of propensity for harm to the fetus. The relevance to the human is not known.

There is no experience in pregnant women who have received this drug.

Precautions. Minor transient elevations of SGOT have occurred in a small percentage of patients. Therefore, this drug should be used with caution in patients with pre-existing liver dysfunction.

Adverse Reactions. The most frequently encountered adverse reactions are related to the gastrointestinal system.

Gastrointestinal and hepatic reactions: anorexia, nausea, vomiting, gastralgia, abdominal cramps, diarrhea and tenesmus, transient elevation of SGOT.

CNS reactions: headache, dizziness, drowsiness, and insomnia.

Skin reactions: rashes.

Dosage and Administration. *Children and Adults:* Antiminth Oral Suspension (50 mg. of pyrantel base/ml.) should be administered in a single dose of 11 mg of pyrantel base per kg of body weight (or 5 mg /lb.); maximum total dose 1 gram. This corresponds to a simplified dosage regimen of 1 cc. of Antiminth per 10 lbs. of body weight. (One teaspoonful = 5 cc.)

Antiminth (pyrantel pamoate) Oral Suspension may be administered without regard to ingestion of food or time of day; and purging is not necessary prior to, during, or after therapy. It may be taken with milk or fruit juices. Because of limited data on repeated doses, no recommendations can be made

How Supplied. Antiminth is available as a pleasant tasting caramel-flavored suspension which contains the equivalent of 50 mg pyrantel base per ml., supplied in 60 cc. bottles.

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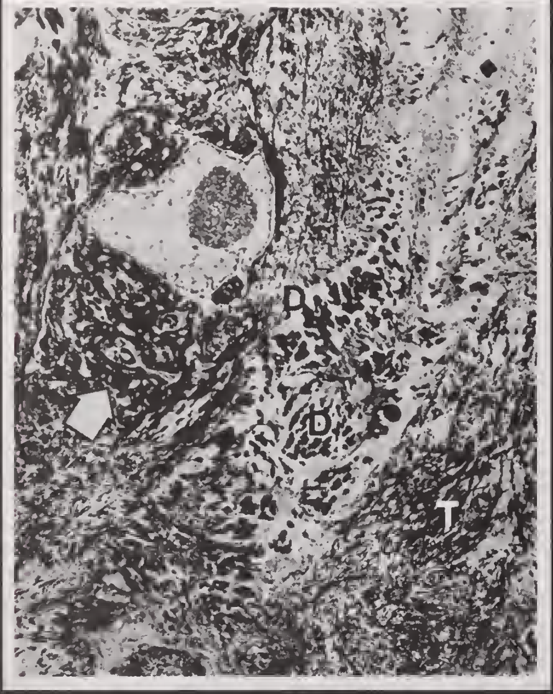
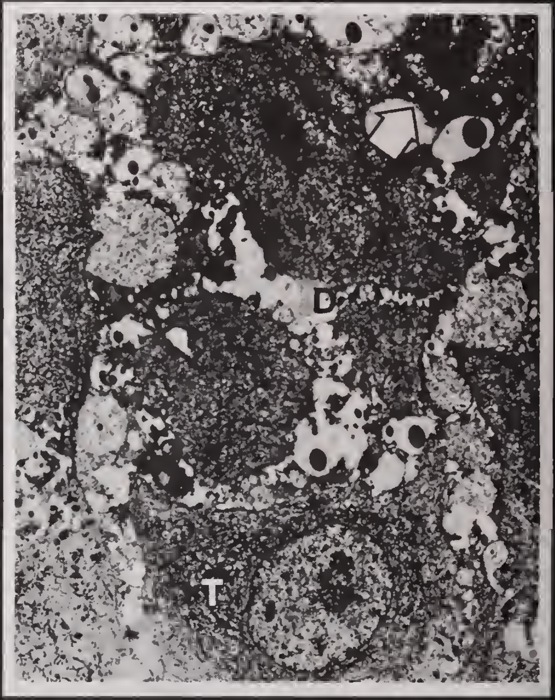


Lesion #2—Two days after initiation of therapy. Electron micrograph of solar keratotic skin from patient's hand.
Typical abnormalities are:
Malpighian cells [containing an abundance of thick tonofibrils (T)] which are connected with well-developed desmosomes (D). Note the clumped tonofibrils in the so-called 'dyskeratotic' cell (arrow) indicative of solar keratosis. No change can be noted at this level after two days of therapy. × 5000 (12/16/71)



Lesion #3—Two weeks after initiation of therapy. Electron micrograph of skin from patient's hand.
Improvement shown:
Less conspicuous desmosomes (D), widened intercellular spaces and Malpighian cells showing a remarkable reduction of tonofibrils (T). The arrow indicates a degenerating dyskeratotic cell. × 5000 (12/31/71)

Solar, actinic or senile keratoses

By whatever name they may be known, they commonly occur as multiple lesions and chiefly on the exposed portions of the skin. Because they may be premalignant, it is generally agreed that they should be treated. Surgery, cryotherapy, or electrodesiccation may present certain drawbacks, both for the physician and the patient, but there is Efudex® (fluorouracil)—as an alternative to conventional therapy.

Sequence of therapy— Selectivity of response

The easily applied Efudex cream or solution usually begins to show effects within a few days—an erythema in the area of the lesions. Within two weeks after initiation of therapy, this reaction usually reaches its height of unsightliness and discomfort, declining after discontinuation of therapy. This reaction occurs in affected areas. Since the response is so predictable, lesions that do not respond should be biopsied to rule out the presence of a frank neoplasm.

Acceptable results

Treatment with Efudex (fluorouracil) provides highly acceptable cosmetic results posttherapeutically. The incidence of scarring is low.* This is particularly important with multiple facial lesions. Efudex should be applied with care near the nose, eyes and mouth.

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*Data on file, Hoffmann-La Roche Inc., Nutley, New Jersey.

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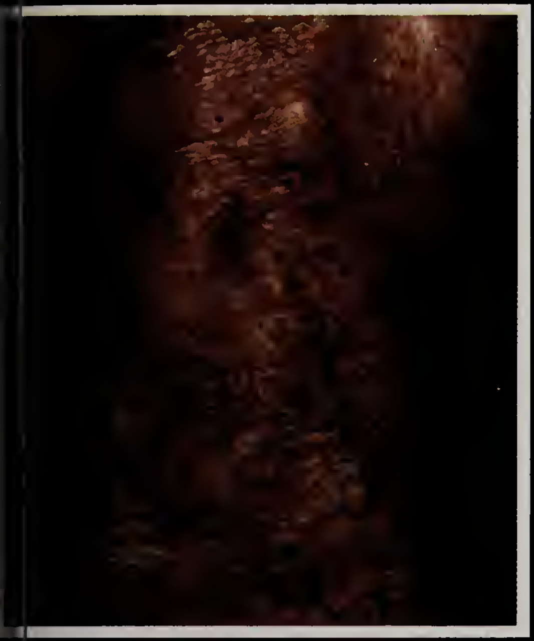
in treating solar keratoses which may be premalignant.



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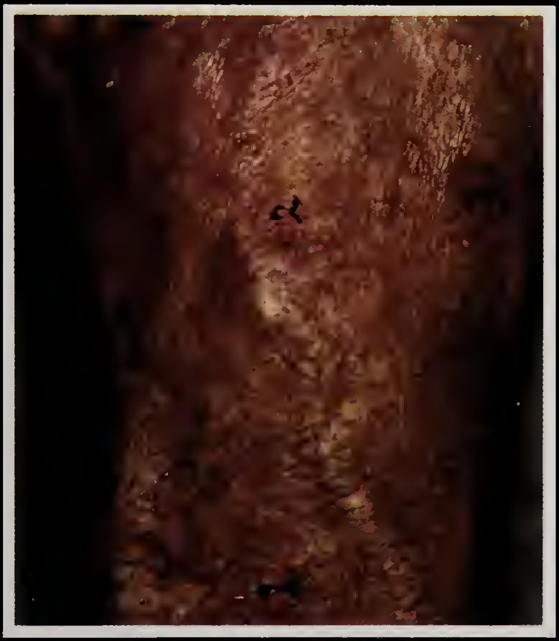

After treatment—Two weeks after therapy stopped—1/28/72

This patient's solar keratoses responded to Efudex (fluorouracil) 5%

Be re prescribing, please consult complete product inf mation, a summary of which follows:

Inc ations: Multiple actinic or solar keratoses.

Co raindications: Patients with known hypersensitivity to an of its components.

Wa ings: If occlusive dressing used, may increase inflam- ma ry reactions in adjacent normal skin. Avoid prolonged ex sure to ultraviolet rays. Safe use in pregnancy not est lished.

Pre autions: If applied with fingers, wash hands immediately. Ap y with care near eyes, nose and mouth. Lesions failing to spond or recurring should be biopsied.

Ad rse Reactions: Local—pain, pruritus, hyperpigmentation an burning at application site most frequent; also derma- titi scarring, soreness and tenderness. Also reported—in- so nia, stomatitis, suppuration, scaling, swelling, irritability, me cinal taste, photosensitivity, lacrimation, leukocytosis, thr mbocytopenia, toxic granulation and eosinophilia.

Do ge and Administration: Apply sufficient quantity to cover les n twice daily with nonmetal applicator or suitable glove. Us l duration of therapy is 2 to 4 weeks.

Ho Supplied: Solution, 10-ml drop dispensers—containing 2% r 5% fluorouracil on a weight/weight basis, com- po ded with propylene glycol, tris(hydroxymethyl)amino- me ane, hydroxypropyl cellulose, parabens (methyl and propyl) and disodium edetate.

Cream, 25-Gm tubes—containing 5% fluorouracil in a vanishing cream base consisting of white petrolatum, stearyl alcohol, propylene glycol, polysorbate 60 and parabens (methyl and propyl).

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Infections Caused by Serratia Marcescens

HABIB O. FARHOUDI, M.D.*

Serratia marcescens, which was first described by the Italian pharmacist Bizio[1] in 1823 was considered to be non-pathogenic for a long time.[2] Woodward and Clark[3] in 1913 described the first case of infection caused by this bacterium. Their patient had pulmonary infection with red sputum which was caused by red pigment of the chromogenic bacteria. Since 1960 there have been an increasing number of reported infections caused by *Serratia marcescens* in the literature. It is a motile gram-negative, aerobic, non-sporulating bacillus belonging to the family of Enterobacteriaceae.[4] It can be identified in the microbiology laboratory on the basis of acid reaction on triple sugar iron-slant and butt with little or no gas in 24 hours, positive Voges-Proskauer, positive Simmon's citrate, inability to ferment lactose, and production of extracellular deoxyribonuclease.[5,6] Most of the strains are non-chromogenic, and only a small % produce a red pigment.

It should be emphasized that the *Serratia marcescens* does not usually produce infection. It is pathogenic only when the host defense mechanism is altered or there have been respiratory and urinary-tract manipulations.[7,8] Nosocomial outbreak due to contamination of nebulizers and inhalation therapy medications have been reported.[9,10] The bacterium has been held responsible for bronchitis, meningitis following lumbar puncture, pneumonia, wound infection, osteomyelitis, lung abscess, arthritis, and, most important, fatal septicemia and endocarditis.[11-14] Severe and fatal infections usually occur in patients with chronic debilitating disorders and especially those with predisposing factors. It has been shown by Dodson[13] that the majority of the patients with septicemia had one or more predisposing factors, such as prolonged treatment with broad-spectrum antibiotics, corticosteroid therapy, mechanical respiratory manipulation, instrumentation of the urinary tract, and particularly cardiac surgery. In a review of 13 known cases of *Serratia marcescens* endocarditis I found that the majority of these patients had cardiac surgery and prolonged multiple antibiotic treatment and that the infection occurred on the prosthetic valve.[15] Three patients, including my case, were heroin addicts. This is the first reported case of pulmonary valve endocarditis by this bacterium. Even with the remarkable increase in gram-negative endocarditis in heroin addicts in the past few years one should remember, that *Staphyloccus aureus* coagulase-positive still remains the most common cause of right-sided endocarditis in this group of patients.[16-18]

The mortality from *Serratia marcescens* septicemia and endocarditis is extremely high. It ranges between 40 and 80%.[12,14] Altemeier et al[14] reviewed 42 patients with *Serratia marcescens* septicemia from the Cincinnati Medical Center, Department of Surgery, and reported a mortality rate of 40%. *Serratia marcescens* endocarditis has an even worse prognosis. From 13 reported cases in the literature only three patients survived.[15] One patient most probably had septicemia and not endocarditis because his blood cultures became negative shortly after removal of the needle and catheter from his vein through which he was receiving heparin. He had no other evidence of endocarditis.[19] The second survivor was cured surgically by the debridément of the tricuspid valve and prolonged antibiotic therapy.[20] The third patient is currently under treatment at the District of Columbia General Hospital. The patient is receiving a combination of Gentamicin and Carbenicillin. One week following this treatment his blood cultures have become sterile.

The treatment of severe *Serratia marcescens* infection appears to be very difficult because this bacterium is resistant to most antibiotics. Studies in our laboratory, consistent with others, show that the organism is usually sensitive to Kanamycin, Chloramphenicol, Gentamicin, and at times Carbenicillin.[11,12,21] Gentamicin seems to be the antibiotic of choice. In my patient the combination of Gentamicin and Carbenicillin was given because these two antibiotics had *in vitro* synergistic action against the organism. Antibiotic tube-dilution studies should be done and the serocidal level of the antibiotic should be determined in an infectious-disease laboratory in order to assure that the adequate level of antibiotic has been attained in the patient's blood. It must be emphasized that cultures from the tip of the intravenous or urinary

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* Assistant Professor of Medicine and Director of the Infectious Disease Laboratory, Howard University Service, D.C. General Hospital, Washington, D.C.

Reprint requests may be addressed to Dr. Farhoudi at the Howard University Service, Department of Medicine, D.C. General Hospital, 19th and C Streets, S.E., Washington, D.C. 20003.

catheter, surgical wounds, inhalation-therapy equipment and sputum should be obtained to determine the port of entry of the bacterium into the blood stream.

Finally, with increasing frequency of gram-negative infections and infections caused by *Serratia marcescens* I hope that clinicians will become more aware of the potential pathogenecity of this bacterium in the comprised host and that this brief communication has served its purpose.

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Comparative Use of Alcohol and Chlorpromazine by Experimentally Stressed CF/1 Mice

RAY B. SMITH, PH.D.*, DENNIS BALCHIUS, B.A.† AND MARY MUELLER, B.A.‡

The use of tranquilizing drugs has all but revolutionized the treatment of alcoholism according to Block.[1] Psychoactive drugs used earlier in the treatment of alcoholism included reserpine, chlorpromazine, promazine hydrochloride and meprobamate. Chlorpromazine was the first of these drugs to be tested on alcoholics in the acute stage according to Mann.[2] The results of such studies caused a near sensation when a synergistic sedation effect was found between chlorpromazine and alcohol. This obviated the continued use of barbiturates and other habit forming sedatives, especially in the treatment of the acute phase of intoxication.

The earlier psychoactive drugs have been joined more recently by numerous newer drugs.[3] From the earlier sedative effect sought in the acute intoxication phase there is now a tendency toward maintenance use of these drugs over longer periods of time.[4] Such use stems from the assumption widely held that alcoholism is a psychogenic problem—a defense against stress.[5] A maintainence use of tranquilizers would presumably keep stress levels from rising above some "drinking threshold" and the alcoholic could go through life leaning on the tranquilizer instead of alcohol, a much more adaptive relationship, psychosocially.

Little research has been done comparing the effectiveness of tranquilizers as a replacement for alcohol in the stressed individual. Casey[6] studied the intake of reserpine and alcohol in rats subjected to electric shock. His subjects increased their intake of alcohol with no change in the intake of reserpine.

There are many difficulties inherent in this kind of experimentation, but the present experimenters accept them, along with the following assumptions: 1. mice will select among available drugs to obtain those that bring the system back toward homeostasis after stress is applied, and 2. a solution that is differentially preferred in a stress situation as compared with a non-stress situation has homeostatic balancing activity.

The drugs involved in the present experiment are chlorpromazine and ethanol. The hypothesis to be tested is that the intake of chlorpromazine, alcohol and water will not differ in experimental subjects prior to and subsequent to stress when compared with controls. Chlorpromazine was chosen for our study because of Goldman's findings that it is more effective than reserpine and has fewer side-effects that might be negatively reinforcing.[7] Further, chlorpromazine has wider use in our Center than does reserpine.

Method

The subjects (Ss) were 28 naive, male mice of the CF/1 strain. All Ss were 19 weeks old.

A stress chamber was used consisting of a large metal container containing light and noise stressers, as used in the experimental psychology department at the State University of New York at Courtland. The effectiveness of this device as a stresser has been reported.[8]

A chlorpromazine solution (0.000125mg/ml H_2O) and an ethanol solution (10% V/V) were available in leakproof water bottles, as was water ad lib. Purina lab-chow was available ad lib throughout the study. These concentrations of solution had been calibrated in a pilot project in terms of equal preference by naive CF/1 mice in the presence of water.

The Ss were separated into two groups of 14 each. For four days both groups were kept on food and water ad lib. The fifth day and for each of the succeding three days group II was stressed in the stress chamber while group I served as controls.

Beginning on the first stress day and continuing throughout the remaining three days of the experiment alcohol and chlorpromazine solutions, along with tap water were available ad lib to both groups. Bottle positions were varied randomly each day.

The amount of each liquid drunk over each 24-hour period was measured for each group, and the mean liquid consumed from each bottle per S was calculated from this overall total. Statistical summaries were computed on the mean consumption per S figure across the four days under the assumption that all but systematic variance would randomize out within groups.

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Results

Total fluid intake was greater during all four days for the stress group when compared with the controls, as indicated in table 1. The consumption of both water and alcohol increased significantly (p 0.001), while that of chlorpromazine showed no difference.

Table 2 gives the percentages of each liquid drunk by each group of *S*s during the four days of stress. The mean percentage of chlorpromazine solution ingested was less for the stressed subjects each day when compared with that ingested by the controls. After the second day the percentage of chlorpromazine solution ingested decreased markedly in the stressed Ss. The difference in total fluid consumption was made up by an increase in the percentage of water consumption.

The probability of the chlorpromazine comprising a smaller percentage and of alcohol comprising a larger percentage of liquids consumed each day for four days is less than 0.003. Thus, the stressed Ss in this experiment increased the relative consumption of alcohol during four days of stress while decreasing the chlorpromazine percentage intake in favor of water.

Unlike in the pilot study the control Ss indicated a preference for the alcohol solution over the chlorpromazine solution every day but one (p 0.05).

Discussion

A determination of the psychoactive concentration of chlorpromazine for

TABLE 1: Liquid Drunk During Four Days of Stress, In Milliliters

Liquid	Stress Day – ml. 1	2	3	4	Average ml. all Days
			Control Subjects		
Water	9.12	9.22	10.12	10.24	9.68
Chlorpromazine	1.70	1.90	1.30	1.50	1.60
Ethanol	2.10	1.90	2.00	2.00	2.00
Total Fluid*	12.92	13.02	13.42	13.74	13.27
			Stressed Subjects		
Water	11.31	11.10	11.38	11.62	11.35†
Chlorpromazine	1.90	2.20	1.10	0.90	1.52‡
Ethanol	3.20	3.60	3.00	3.40	3.30†
Total Fluid*	16.41	16.90	15.48	15.92	16.18†

* The average water consumption over each of four days preceding the experiment was 11.4 ml and 11.2 ml for the controls and stressed subjects repectively.

† Difference between control and stressed Ss significant beyond 0.001 level of confidence.

‡ No difference between the two groups.

TABLE 2: Liquid Drunk During Four Days of Stress In Percentage of Total Liquid Drunk

Liquid	Stress Day – ml 1	2	3	4	Average ml. all Days
			Control Subjects		
Water	70.59	70.82	75.41	74.53	72.83
Chlorpromazine	13.16	14.59	9.69	10.92	12.10
Ethanol	16.25	14.59	14.90	14.55	15.07
Total	100.00	100.00	100.00	100.00	100.00
			Stressed Subjects		
Water	68.92	65.68	73.51	72.99	70.27
Chlorpromazine	11.58	13.02	7.11	5.65	9.34*
Ethanol	19.50	21.30	19.38	21.36	20.39†
Total	100.00	100.00	100.00	100.00	100.00

* The probability of obtaining a smaller percentage of Chlorpromazine among stressed subjects, 4 days in a row, is less than 0.003.

† The probability of obtaining a larger percentage of Ethanol among stressed subjects, 4 days in a row, is less than 0.003.

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the CF/1 mouse has not been found in the literature, and an earlier pilot study calibration of ethanol and chlorpromazine solutions, as measured by similar ingestion rates by the naive CF/1 mouse, did not hold up in the present study. Neither the total amounts of chlorpromazine solution and alcohol solution ingested, nor the percentages of each in the total fluid intake were markedly disparate among the control Ss.

That the total fluid intake increased among controls, once chlorpromazine was offered, is in keeping with earlier studies.[9] The increase in total fluid intake among stressed subjects also has precedence in the literature.[10]

The present study indicated that chlorpromazine intake in this concentration was not increased by stressed Ss, while mice chose significantly increased amounts of alcohol. The fact that the percentage of chlorpromazine ingested by stressed Ss gradually decreased and was always at a smaller percentage of total fluid intake than that of the controls suggests that some effect was operating and discernable by the stressed Ss. In any event the mice did not increase their consumption of chlorpromazine in the presence of stress and certainly not in preference to alcohol.

These results, while not intended to be generalized to the human population of alcoholics, bear out the experiences of many of our therapists with the use of chlorpromazine and other psychoactive drugs with alcoholics, especially where stress was observed and alcohol available. Even nonalcoholic persons have been observed to choose alcohol over psychoactive drugs in these situations. The implication is that alcohol has a qualitatively or quantitatively different effect in re-establishing homeostasis in the presence of stressers than do the psychoactive drugs currently used. A more interesting implication is that the psychogenic theories of alcoholism may need revision. That is, that the biochemical effects esoteric to alcohol other than purely sedative action may be responsible for its wide use in stress situations plus a multitude of other situations.

Finally, the question should again be raised as to the relative catabolic effects and overall effectiveness of the use of alcohol vs. tranquilizers in ameliorating or controlling the acute phases of withdrawal.

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Flucytosine* Therapy in Cryptococcosis. Report of a Case.†

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A newly developed drug, Flucytosine (Ancobon), was successfully used to treat a 6½-year-old child with disseminated *Cryptococcal neoformans* infection. Amphotericin B, since its discovery in 1956, has been the main therapeutic agent for all forms of cryptococcal diseases. The therapy with amphotericin B manifested renal toxicity in virtually all cases with elevation of blood urea nitrogen and lowering of creatinine clearance. In some case reports, 85% of the cases treated with amphotericin B showed irreversible renal damage. There was also 10-15% relapse in meningeal cryptococcosis.[1] Grunberg and associates[2] reported their studies in 1964 of Flucytosine therapy in mice with candida and *Cryptococcus neoformans* infection. This report was followed by Tassel and Maddoff,[3] and Utz et al,[1] which showed encouraging results of Flucytosine therapy in adults with cryptococcal infections. Recently Flucytosine has been used in children with cryptococcosis with satisfactory results.[4,5]

Flucytosine is a cytosine, a fluorinated pyrimidine chemically related to fluorouracil and flaxuridine. It is a white crystalline powder, odorless with molecular weight 129.1. It is fairly soluble in water (1.5% at 25C°). This substance has *in vitro* and *in vivo* activity against candida and cryptococcus. The exact mode of action against these fungi is not known.

Following is a case report of 6½-year-old boy which was treated at D. C. General Hospital for cryptococcosis. This patient has survived and shown no evidence of disease after more than 20 months following treatment.

Report of Case

A 6½-year-old male Negro was admitted with complaints of general malaise, weight-loss and low-grade fever of two months duration. The child had been healthy prior to this illness. He was noticed to have anorexia and easy fatigability. He became anorexic and later began to vomit. A weight loss of 6-7 pounds was reported. One week prior to hospitalization he appeared to be markedly drowsy, falling asleep during daytime. He also complained of abdominal pain and distension. There was no concomitant history of chronic cough, jaundice, abnormal urination, bone pains, or bleeding tendency.

The birth history was unremarkable. He was full-term infant born by spontaneous vaginal delivery. His birth weight was 9 pounds and 3 ounces. The neonatal and infancy period was uneventful. He sat up at the age of 6 months, talked at 12 months, and walked at 15 months of age.

There had been no major illnesses. One brother had bacterial meningitis two years before and was treated and survived.

The boy was listless, pale, lethargic, fretful, and moderately dehydrated. His weight was 37 pounds (10th percentile), temperature 101°F, pulse 130/minute, and respiratory rate 35/minute. Examination of the head, ear, nose and throat, chest, cardiovascular system, and skin revealed normal findings. The abdomen was distended; the liver was palpable 10 cm below the right costal margin, the surface was smooth, firm, and nontender on palpation, with a sharp and well-defined edge. The splenic tip was palpable. There was free fluid demonstrable in the abdomen. The genitalia were normal, and no hernia was apparent. The pupils were equal and reactive to light with no extra ocular movements. The fundi were normal and the cranial nerves intact. The sensory and motor functions were intact. Babinski's sign was negative. There was no nuchal rigidity, and Kernig's and Brudzinski's signs were absent.

Initial laboratory studies showed hematocrit 28%; sedimentation rate 69 mm 1st hour; WBC 14,000, polymorphs 79%, lymphocytes 9%, and basophils 12%; platelets 726,000; reticulocytes 1.1%. Sickle-cell prep was negative, and hemoglobin electrophoresis showed AA pattern. Serology for syphilis was negative. Glucose-6-phosphate dehydrogenase was normal. Chemistries: BUN 7 mg%, glucose 112 mg%, Na 136 mEq/L, K 3.4 mEq/L, Cl

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* The Flucytosine (Ancobon) used in this investigation was supplied as RO 2-9915 by Donald C. Carlton, M.D. of Hoffmann-LaRoche, Inc., Nutley, New Jersey.

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90 mEq/L, and CO2 31 mM/L. Urinalysis was within normal limits.

The child was given intravenous fluids 0.25% NaCl in 5% dextrose solution for 48 hours. He then tolerated oral fluids. During this period he continued to have temperatures ranging up to 103°F. These were controlled by aspirin. Cultures from the throat, nasopharynx, stool, and blood were taken. He was then empirically placed on 2.0 gm Ampicillin intravenous daily. On the 4th hospital day he was noticed to be lethargic and semi-conscious. He developed nuchal rigidity, but there were no other changes in the physical examination.

A spinal tap was done, and showed a clear, colorless fluid, with free flow of cerebrospinal fluid. Gram stain was negative. Microscopic examination showed 60 round and oval cells of different sizes and with budding. These were consistent with yeast cells. The sugar was less than 10 mg%, and the protein was over 100 mg%. The cerebrospinal fluid was cultured on Sabourand's medium and in 24 hours grew a thick layer of mucoid creamy growth. Organisms resembling cryptococci were seen by the use of India Ink preparation (Fig. I). These were confirmed by a positive urea test.

The diagnosis of cryptococcal meningitis was made, and the patient was started on Amphotericin B therapy, 0.5 mg/Kg each 24 hours. Cultures of blood, throat, urine and stool were negative for pathogenic bacteria. Roentgenograms of the chest, abdomen, skull and long bones were negative. Further evidence of *Cryptococcus neoformans* infection was obtained from a liver biopsy which showed granulomatous lesions with the presence of numerous cryptococci (Fig. 2).

The bone marrow smear and biopsy, however, were normal and did not reveal any organism or granulomatous lesions. Skin tests for tuberculosis, histoplasmosis and coccidiomycosis were negative. An intravenous excretory urogram was reported normal.

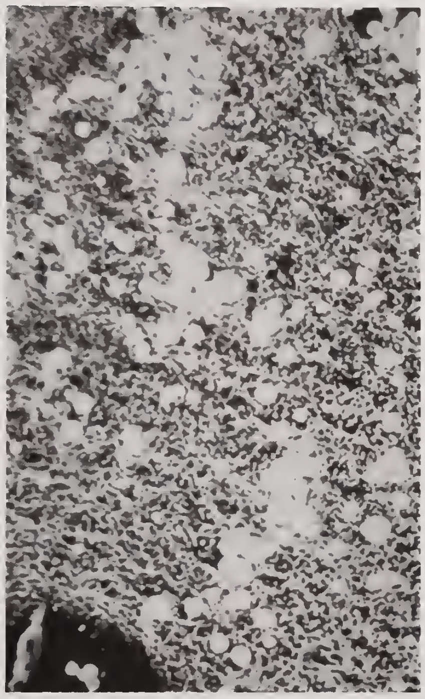

FIG. 1. India ink preparation of cerebral spinal fluid showing numerous *Cryptococcus neoformans.* Note budding cells.

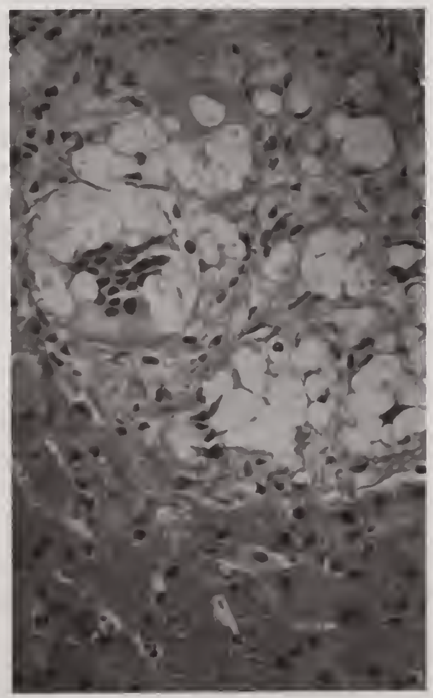

FIG. 2. Liver biopsy. Note encapsulated clusters of *Cryptococcus neoformans* in parenchyma.

Amphotericin B was increased to 1.5 mg/Kg each 24 hours intravenously and intrathecally on the second day of therapy. This was further increased to 2.5 mg/Kg each 24 hours intravenously and 0.6 mg intrathecally on the third day (table 1). The boy was given hydrocortisone 100 mg daily from the second day of amphotericin B therapy in order to decrease the inflammatory reaction in the central nervous system.

The child became afebrile and alert following the 1st week of therapy. He began to respond to hospital personnel and parents during the second week. He started to feed himself, sat up, and expressed his needs by the end of the third week.

After two months of Amphotericin B therapy the child was afebrile, alert, and had a good appetite. The spinal fluid still showed 1-2 *Cryptococcus neoformans*/PHF, but culture on Sabouraud's media failed to grow. The intrathecal amphotericin B was then reduced to once weekly.

In view of the elevation of BUN, albuminuria, persistence of cryptococci in the spinal fluid, and the failure of the organisms to grow on Sabouraud's media, the patient was given Flucytosine 750 mg every 6 hours orally for 30 days. He did not show any alteration in temperature and maintained clinical improvement. There were no adverse reactions as shown in blood chemistries and hematologic studies. There was a significant lowering of BUN. Spinal taps performed during Flucytosine therapy revealed no visible cryptococci in the spinal fluid and no growth.

The patient was discharged on the 20th day after discontinuation of Flucytosine therapy. Since that time he has been followed on an outpatient basis.

A psychological evaluation following therapy showed that the patient's I.Q. varied between 40-48. It was the examiner's impression that the patient was functioning at borderline level and would require special schooling. An

TABLE I: Schedule and duration of medication received by this 6-1/2-year-old boy with proven disseminated *Cryptococcal neoformans* infection

Medication	Daily Dose	Date Started	Date Completed
Ampicillin	2.0 gm	3/11/70	6/13/70
Ampicillin	2.4 gm	4/22/70	5/4/70
Amphotericin B	11mg	3/8/70	3/9/70
Amphotericin B	18mg	3/9/70	3/10/70
Amphotericin B	30mg	3/10/70	6/11/70
Hydrocortisone	100mg	3/11/70	6/13/70
Flucytosine	3.0 gm	6/11/70	7/11/70

ophthalmological consultation following therapy showed bilateral optic atrophy, greater in the right eye, and multiple, apparently inactive, chorioretinal lesions in both eyes. It was thought that these lesions were secondary to hematogenous spread of the cryptococci and not the result of therapy.

Follow-up visits showed the patient to be in good health, with no hepatosplenomegaly, although there was reduced mental capacity and reduced vision.

Discussion

Among the major sources of *Cryptococcus neoformans* is pigeons excreta and to a lesser extent excreta of other birds. Cryptococcal infections often start from the lungs and later spread to other organs through the blood stream. The disease becomes chronic after an insidious onset, often with a fatal outcome, although remissions have been described without treatment. Pulmonary lesions may be multiple. There may be hematogenous spread to the liver, spleen, kidneys, brain, and other organs. The reactions are usually chronic. Tubercle-like lesions have been described. Lesions of bone in the form of solitary osteomyelitis may occur. The central nervous system manifestations are meningitis, meningoencephalitis, and occasionally granulomatous lesions simulating space-occupying tumors.

Amphotericin B has improved the prognosis of cryptococcal infections but has some disadvantages. It is given intravenously and has serious adverse reactions.[1,4] Flucytosine is less toxic, can be given orally, and has minimal side-effects. The course of Flucytosine varies from 3-10 weeks depending upon clinical improvement.[4,5]

In our patient cryptococci persisted in the spinal fluid, without growth on Sabourand's media while on Amphotericin B therapy. It was completely eradicated with Flucytosine therapy. There were no manifestations of toxicity as monitored by measurements of glutamic-oxaloacetic transaminases, alkaline phosphatase, lactic dehydrogenase, BUN, WBC, and hematocrit. The BUN fell during Flucytosine therapy.

The reported side-effects of Flucytosine include nausea, vomiting, diarrhea, and rash. There are reports of hepatotoxicity in 10% of the patients treated, but this is reversible. Other toxic effects include pancytopenea but are not well documented.[6] Our patient did not manifest any evidence of toxicity to Flucytosine.

At present Flucytosine appears to be less toxic and more efficacious, and may ultimately prove to be drug of choice in all cases of cryptococcal infections.

Acknowledgments

The authors wish to express their appreciation for Dr. Sophie Perry for interpretations of liver biopsy and Dr. C. K. Lee for the initial work-up of the case.

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Hodgkin's Disease Terminating in Acute Erythromyeloblastic Leukemia with Diabetes Insipidus.

A Case Report and Review.*

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The association of acute leukemia and Hodgkin's disease is rare. Thirteen cases have been described in the English language literature[1–8] of acute leukemia in various forms complicating Hodgkin's disease. The following is a case of acute erythromyeloblastic leukemia with onset 10 years after the diagnosis of Hodgkin's disease was made.

Report of Case

R. H., a 23-year-old white man, was evaluated in 1956 for painless right axillary lymphadenopathy. Biopsy revealed Hodgkin's disease. In 1958 he had recurrence in the right axilla and extension to the right cervical region; he was treated with 3,000 R. and 5,000 R. to these areas, respectively. In March 1960 he was discovered to have left cervical, supraclavicular, and bilateral inguino-femoral lymphadenopathy, and hepatomegaly. In addition, an excretory urogram revealed a displaced right ureter, suggestive of enlarged retroperitoneal nodes. He was given 1,800 R. to the left side of the neck and a course of nitrogen mustard. In June 1961 he was discovered to have recurrence of bilateral inguinal adenopathy and was again treated with a course of nitrogen mustard. In 1962, because of an increase in the size of the spleen further nitrogen mustard injections were given, and the patient was placed on maintenance chlorambucil, which was continued off and on until March 1965. In 1964 he received radiation therapy to both femoral-iliac areas because of lymphadenopathy. He was first seen at Walter Reed General Hospital in July 1964, at which time there were no palpable lymph nodes, spleen, or liver.

In early 1965 he was noted to have gradually falling white blood cell, red blood cell and platelet counts to low values of WBC 3500/$mm.^3$, Hct 27%, and platelets 50,000/$mm.^3$ A bone-marrow aspirate at that time was normocellular with adequate numbers of megakaryocytes and an M:E ratio of 5:1. There was normoblastic maturation of the erythrocytic precursors. Granulocytic maturation was normal and orderly. Approximately 7% plasma cells and 15% mature lymphocytes were noted. A Cr^{51} RBC survival study was done, revealing a half-life of 16 days with a predicted mean of 18 days. Because it was thought that a hypersplenic state was contributing to the patient's pancytopenia, a splenectomy was performed on August 23, 1965. The spleen weighed 579 gm but was otherwise unremarkable. Microscopically, prominent, non-caseating granulomata and numerous Reed-Sternberg cells were seen. An accessory spleen was also removed at the time of operation. By August 30, 1965 the patient's WBC count had risen to 8,200/$mm.^3$, Hct to 35%, and platelet count to 200,000/$mm.^3$, with a reticulocyte count of 2.2%.

On September 29, 1965 inferior vena cavography was done, revealing slight displacement of the vein anteriorly and laterally, suggestive of enlarged retroperitoneal nodes. In the latter part of 1965 the patient was given several courses of chemotherapy, including prednisone, cyclophosphamide, chlorambucil, and vinblastine for recurrent generalized lymphadenopathy.

Beginning in March 1966 several nucleated red blood cells were noted on peripheral smears. A CBC on 11 May 1966 revealed: WBC 14,800/$mm.^3$, Hct 37.5%, platelet count 127,000/mm^3, and 60 nucleated RBC's/100 WBC's. CBC on 22 June 1966 revealed WBC 61,800/$mm.^3$, Hct 33.5%, platelet count 84,000/$mm.^3$, with a reticulocyte count of 6.8% and 400 nucleated RBC's/100 WBC's. The red blood cells on the peripheral smear were

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normochromic and macrocytic with marked aniso- and poikilocytosis; a few target cells and Howell-Jolly bodies were also noted. The nucleated red blood cells showed some dysynchrony of nuclear-cytoplasmic maturation. A WBC differential count revealed 41% myeloblasts, some with Auer rods (Fig. 1), 2% promyelocytes, 1% myelocytes, 10% bands, 26% segmented neutrophils, 15% lymphocytes, and 5% monocytes. Bone-marrow aspirate (Fig. 2) revealed a hypercellular specimen with decreased numbers of megakaryocytes. The M:E ratio was 2:1. There was a pronounced left shift in the granulocytic series with an obvious leukemic hiatus. Approximately 55% of the cells were myeloblasts. Erythroid precursors demonstrated moderate megaloblastosis. An occasional giant metamyelocyte and band form were seen. The diagnosis of acute erythromyeloblastic leukemia was made. Cytogenetic studies performed of the bone marrow revealed a mosaic pattern of 46, XY/47,XY,C^+.

The patient was treated with prednisone and 6-mercaptopurine with some improvement. Because of pain in the mid back and right shoulder, a bone scan was done, which revealed a slight increase in uptake in the vertebral bodies of T-6, 7, 8, 10 and 11 and in the right shoulder. There was also increased uptake on the bone scans of the manubrium and left sacro-iliac joint. In November 1966 an emergency appendectomy was performed, which revealed an acute purulent appendicitis; microscopic examination revealed no evidence of Hodgkin's disease or leukemic infiltration.

In January 1967 the patient began to complain of pain in the right hemithorax. A chest X-ray film revealed a posterior mediastinal mass, for which the patient received 2,200 R. to the area; therapy was interrupted because of the development of thrombocytopenia and was not resumed. A 90% shrinkage of the tumor was noted, with relief of pain.

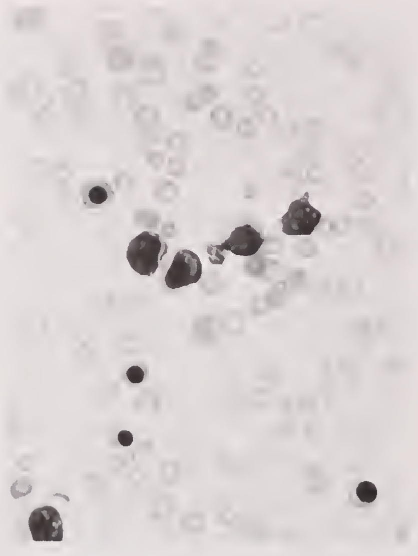
FIG. 1. Peripheral blood revealing nucleated red blood cells and myeloblasts. An Auer rod can be seen.

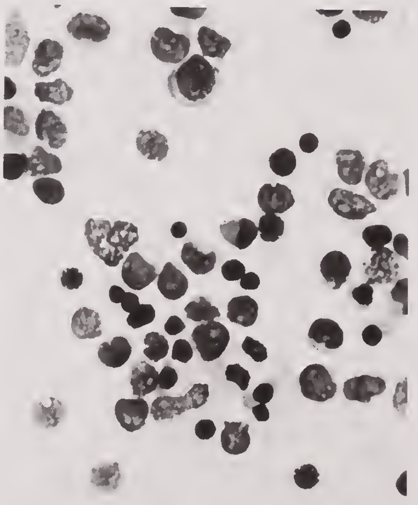
FIG. 2. Bone marrow aspirate showing early granulocytic forms and megaloblastic red blood cell precursors.

In March 1967 the patient was noted to be failing on 6-mercaptopurine and prednisone and so was given several five-day courses of prednisone, vincristine, methotrexate and 6-mercaptopurine (a modified POMP regimen), with resulting marked decrease in size of enlarged lymph nodes and decline in the peripheral WBC count, although myeloblasts and nucleated RBC's remained in the peripheral smear. At about this time, the patient developed diabetes insipidus and was treated with long-acting pitressin intramuscularly with fair control. Also in March 1967, because of an enlarging mediastinal mass, the patient was given 800 R. to the midplane of the mediastinum; this course was interrupted because of the patient's general deterioration and was not resumed.

On June 26, 1967 the patient was admitted to the hospital with the chief complaint of right upper quadrant pain and appeared extremely ill. He was found to have a left pleural effusion, and thoracentesis produced 600 ml of a cloudy, yellow fluid. He was given kanamycin, cephalosporin, and colistin intravenously but became progressively more dyspneic and died five hours later.

Autopsy revealed a left hemothorax, with the left pleural space containing 1,200 ml of blood. Both lungs contained numerous tumor nodules which contained Reed-Sternberg cells and were classified as Hodgkin's disease, nodular sclerosing type. The bone marrow was very hypercellular with an increased fibrous stroma. There was an increase of immature elements of both the myelocytic and erythrocytic series, and rare Reed-Sternberg cells were also seen in the bone marrow. Mediastinal and retroperitoneal lymph nodes were replaced by leukemic cells. There were also leukemic cells in the liver, heart, and kidneys. The brain was free of involvement.

Discussion

The development of acute leukemia in patients with Hodgkin's disease is rare. Most reported cases fall into the acute myelocytic[1,7,8] or myelomonocytic[3] forms. Acute monocytic,[6] stem-cell,[4] and unspecified[5] varieties have also been described. Only three cases of erythroleukemia[1,2,7] complicating Hodgkin's disease are noted in the English literature. These cases are summarized in table I. Of interest is the fact that our patient manifested a progressive erythroblastic peripheral blood picture at least three months prior to the diagnosis of his acute erythromyeloblastic leukemia. These erythroblasts became increasingly bizarre and immature, suggesting a leukemic process, as opposed to bone marrow involvement with Hodgkin's disease alone.

Diabetes insipidus, diagnosed a few months prior to the patient's death during his leukemic phase, has been reported to occur in several patients with acute and chronic leukemia[9] but has not been reported in patients with Hodgkin's disease alone. Examination of our patient's brain at autopsy failed to reveal leukemic infiltrates or evidence of Hodgkin's disease and was thought to be pathologically normal, although the supraoptico-hypophyseal system was not specifically examined.

Cytogenetic studies in patients with Hodgkin's disease terminating in acute leukemia have often revealed aneuploidy that is either hypodiploid, hyperdiploid, or pseudodiploid (table I). Our patient was found to have in his bone marrow a mosaic pattern of diploid cells and cells with a C trisomy. A supernumerary chromosome belonging to group C has been encountered in malignant and proliferative disorders of myeloid, monocytoid and erythroid, but not lymphoid, cells.[10] Hyperploidy, presumed to represent Reed-Sternberg cells, has been demonstrated in some cases of untreated Hodgkin's disease, but a C trisomy in these patients has not been reported.[11]

All patients in table I had received radiation therapy, instituted nine months[8] to 18 years[3] prior to the diagnosis of leukemia. The median latent period between the first course of radiation and the appearance of leukemia in this reviewed series is five years. Our patient had received radiotherapy for his Hodgkin's disease beginning eight years prior to the diagnosis of erythromyeloblastic leukemia.

Radiation may induce a leukemic state, as clearly demonstrated in patients with ankylosing spondylitis[12,13] treated with radiation and among the atom-bomb survivors,[14] in whom the mean interval between radiation and the onset of leukemia was four to six years. The increased incidence of leukemia in patients with

TABLE I: Reported cases in the English literature of Hodgkin's disease terminating in acute leukemia.

Reference	Age at diagnosis of Hodgkins Disease (H.D.) and cell type if specified	Therapy	Age at Dx of leukemia and type	Comments
Newman[3]	9, white boy paragranuloma	10 courses of radioRx over 16 years to a total of 27,025 R. to 32 ports. Short course of nitrogen mustard in 1966.	28, acute myelomonocytic leukemia	Some response to 6-mercaptopurine. Died 3 months after Dx made. Autopsy not performed.
Newman[3]	25, white man nodular sclerosis	RadioRx on 4 occasions to axillae, neck, mediastinum, and thoracic and lumbar areas.	26, acute myelomonocytic leukemia	Died 2 days after Dx. Autopsy—generalized leukemic infiltrates and involvement with H.D.
Newman[3]	33, white woman nodular sclerosis	8 courses of radioRx: 11675 R. to 19 ports over 6 years; several courses of chemoRx, including nitrogen mustard and chlorambucil.	38, acute myelomonocytic leukemia	Died 1 month after Dx. Autopsy not performed.
Newman[3]	63, white man paragranuloma	RadioRx to an inguinal area.	68, acute myelomonocytic leukemia	Patient under therapy at time of publication of case report.
Steinberg[7]	30, white man	5 courses of radioRx over 8-year period to right cervical, lumbar spine, right lung, left cervical and bilateral inguinal. Several response. courses of chemoRx including nitrogen mustard and chlorambucil.	38 acute myelocytic leukemia	Chromosome studies revealed aneuploidy with breaks, gaps and deletions. ChemoRx with 6-mercaptopurine given without response. Died 6 months after Dx. Autopsy not performed.

(continued)

TABLE I: –*Continued*

TABLE I: Reported cases in the English literature of Hodgkin's disease terminating in acute leukemia.

Reference	Age at diagnosis of Hodgkins Disease (H.D.) and cell type if specified	Therapy	Age at Dx of leukemia and type	Comments
Steinberg[7]	36, white woman	2 courses of radio Rx: 5000 R. to cervical, supraclavicular and mediastinal areas. Several courses of chemoRx, including nitrogen mustard, chlorambucil and vinblastine.	51, acute erythroleukemia	Chromosome studies revealed hypodiploidy and rearrangements. Died 1 month after Dx. Autopsy not performed.
Osta[8]	60, Filipino man lymphohistiocytic	4 courses of radioRx beginning 9 months before Dx of A.L.: 4100 R. to bilateral cervical areas, 3000 R. to mediastinum, 3000 R. to periaortic area, and 3000 R. to midplane to pelvis; short course of vinblastine.	60, acute myelocytic leukemia	Died a few days after Dx. Autopsy revealed generalized involvement with H.D. and leukemic infiltrates.
Ezdinli[1]	33, white woman nodular sclerosis	Several course of radioRx over 7 years: 3034 R. to mediastinum, 1485 R. to left axilla, 2560 R. to each cervical area, 2560 R. to abdomen, 3000 R. to left cervical area; 1 course of chemoRx with nitrogen mustard.	40, acute myelocytic leukemia	Treated with 6-mercaptopurine and methotrexate without response. Chromosome studies revealed hyperploidy. Died 4 months after Dx. Autopsy revealed H.D. and leukemic infiltrations.
Ezdinli[1]	72, male negro lymphocyte predominance	2 courses of radioRx: 3500 R. to left cervical area and 2800 R. to periaortic nodes.	74, acute erythroleukemia	Chromosome studies revealed hyperploidy. Treated with 6-mercaptopurine, prednisone, daunomycin and hydroxyurea without response. Died 5 months after Dx. Autopsy not done.
Durant[2]	37, white man	29 courses of radioRx, between 200-2016 R. to right cervical, bilateral supraclavicular and axillary, mediastinum, left anterior chest, right lower abdomen and pelvis, and left elbow. Several courses of chemoRx, including chlorambucil, cyclophosphamide, vinblastine, and nitrogen mustard.	47, DiGuglielmo's leukemia	Chromosome studies revealed marked aneuploidy. No response to therapy with methylhydrazine, 6-mercaptopurine and prednisone. Died 4 months after Dx. Autopsy revealed extensive leukemic infiltrates but no evidence of H.D.
Lacher[4]	23, white woman granuloma	One course of radioRx: 2000 R. to ant. mediastinum. Several courses of chemoRx, including nitrogen mustard and chlorambucil.	26, stem cell, probably lymphoblastic	No response to Rx with 6-mercaptopurine and prednisone. Died 3 days after Dx. Autopsy revealed generalized involvement with leukemia and H.D.
Cohen[5]	26, white woman	Radical mastectomy age 28 for carcinoma right breast with post-op. radioRx to right anterior chest, right axilla and supraclavicular areas; ovarian irradiation, cervical and left axillary node irradiation.	30, unspecified	Died a few months after Dx.
Razis[6]	27, white woman	Several courses of radioRx	33, acute monocytic	Leukemia discovered at autopsy.

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polycythemia treated with X-irradiation and/or radioactive phosphorus (^{32}P) is well known (20-40 times greater than expected for a comparable population at risk).[15-18] Some patients with polycythemia vera, however, have developed an acute leukemia without a prior history of exposure to X-irradiation, having received chemotherapy[18] or only venesection.[19] Carey et al[20] reported four women with acute myelocytic leukemia developing within 11 months following the diagnosis of carcinoma of the breast in which no radiotherapy had been given. Acute myelocytic leukemia following prolonged melphalan therapy for multiple myeloma has been reported.[21,22] Cases of ovarian[23,24] and lung[25] carcinoma treated with cytotoxic chemotherapy and terminating in acute leukemia have also been reported.

Although many of these cases demonstrate an apparent direct relationship between radiation and the incidence of leukemia, other factors may be important, such as the role of chemotherapy, longer patient survival, and the natural history of the basic disease process. Continued reporting of these cases is necessary to determine possible patterns of development of acute leukemia.

Acknowledgment

The authors wish to thank Mahlon Freeman, Col. MC USA, of the Gynecology and Breast Pathology Section, AFIP, who performed the chromosome analysis on this patient and provided helpful information on cytogenetic studies.

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Ulcerative Disease of the Colon Proximal to a Benign Neoplasm*

MELVYN B. LEWIS, M.D. AND WILLIAM O. CRAWFORD, JR., M.D.

There have been numerous case reports in recent literature describing acute inflammatory changes of the colon proximal to partially obstructing lesions. The entity has been variously described as ulcerative colitis[1,2], necrotizing colitis[3] and pseudomembraneous colitis.[4,5] Despite discrepancy in terminology and minor variation in their description of mucosal patterns the entities apparently are similar in etiology, roentgen appearance, and pathology.

The fact that significant partial obstruction from whatever cause plays a vital etiologic role is exemplified by the variety of causes of obstruction which have been reported in the literature. The largest group has been in patients with adenocarcinoma. This is probably due to the higher incidence of malignancy as a cause for colonic obstruction. The entity, however, has been reported secondary to other forms of chronic obstruction. The enterocolitis occurring proximal to the aganglionic segment of Hirschprung's disease is one of the better known.[6] A similar entity occurs proximal to ileostomy as "prestomal ileitis."[7] Proximal colitis also has been reported in obstructions caused by a surgically produced colonic stricture,[8] radionecrosis following radiation therapy for carcinoma of the cervix, and a pseudotumor of the iliac crest obstructing the rectum in a hemophiliac.[5] To date there have been no reports of colitis occurring proximal to an intraluminal neoplasm which was not malignant.

Report of Case

A 67-year-old white man came to the Georgetown University Medical Center because of increasing flatulence, constipation and passage of bloody mucus from the rectum. He denied having malaise, weight loss, fever, or previous bowel disorder. He used laxatives only occasionally, and Milk of Magnesia was adequate for control of his constipation. Physical examination revealed a healthy looking white man in no distress. No masses were palpable in the abdomen, but there was questionable tenderness in the left lower quadrant. Sigmoidoscopy to 19 cm was negative. The hematocrit was 40.5 with a 6,000 white blood cell count and a normal differential.

A barium enema performed prior to admission (figs. 1-3) showed a 5 cm sessile polypoid intraluminal mass at the junction of the descending and sigmoid colon. The lesion was broad-based, and the surrounding colon showed no evidence of invasion or fixation. The entire colon proximal to the lesion showed generalized diffuse mucosal irregularity. There were no stenotic areas or areas of significant dilatation. Some loss of the normal haustration was noted.

The patient was admitted, and an exploratory laparotomy was performed. A 5 cm firm polypoid mass with some serosal inflammation was found. There was normal mucosa partially covering the lesion, and some superficial ulceration was noted. An anterior resection was performed with 13 cm of colon removed. The proximal colon was not explored but "appeared normal."

The resected specimen showed well differentiated adipose tissue with many vascular structures characteristic of a lipoma. The mucosa covering the mass and around the base showed epithelial hyperplasia. The surgical specimen included 3.5 cm of proximal colon which was "free of disease."

The patient had an uneventful postoperative course and recovery. He was discharged on the eleventh postoperative day.

Discussion

The etiology of inflammatory changes proximal to chronic partially obstructing disease is still obscure. It has been noted that chronic obstruction, regardless of the cause, leads to alterations in the wall of the colon even without coexistent ulcerative mucosal disease. Anatomically one sees muscular hypertrophy, shallow haustrations, and mucosal edema with irregularity.[9] The colon therefore shows underlying physiological changes prior to the development of recognizable ulcerative disease of the mucosa.

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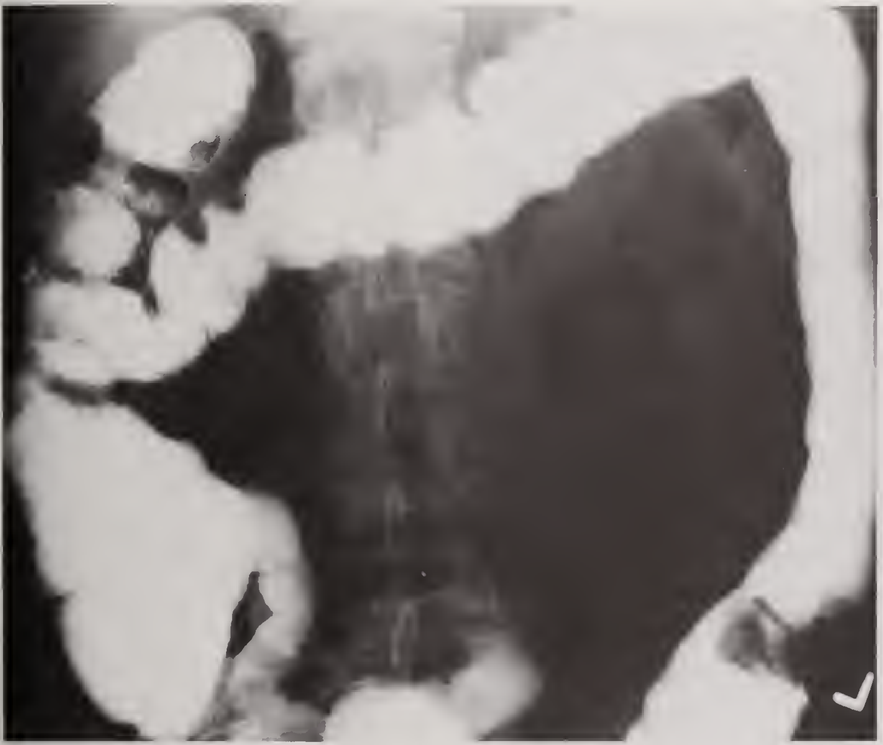

FIG. 1. There is diffuse generalized irregularity of the mucosal surface characteristic of superficial ulceration. The process is seen extending proximal from the lipoma to include the entire mucosal surface including the cecum.

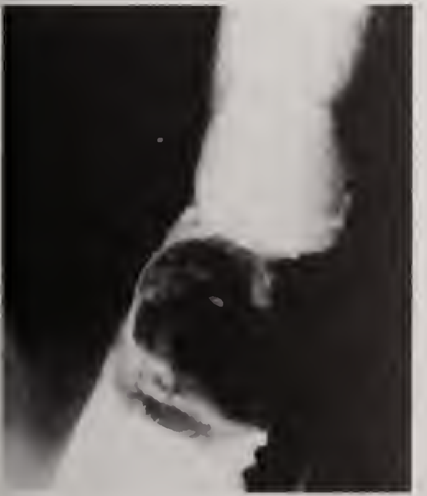

FIG. 2. Spot film of lesion, showing sessile, obstructing nature of the lesion. The difference between the proximal ulcerated and the distal normal mucosa is demonstrated.

In 1966 Gloetzer and Pihl[10] experimentally produced various degrees of obstruction in dogs surgically. They found no qualitative or quantitative changes in fecal flora to explain the possibility of infection as a possible etiologic factor. Various authors have postulated ischemia as a major factor.[1,4,6,10] They argue that the chronic increase in intraluminal pressure results in chronic "stretching" which leads to ischemia and ultimately to mucosal infarction. This might explain the abrupt change from normal to abnormal mucosa that has been a rather constant finding.[11] It has been noted, however, that there is no correlation between the degree of obstruction and the severity and extent of the inflammatory changes.[3] Goulston and McGovern[5] state that the earliest lesion is on the mucosal surface and only later advances into deeper layers. They conclude that either an intraluminal or intramucosal toxic agent, acting locally, is a more likely explanation. This may well explain their single case report of ulcerative disease distal as well as proximal to an obstructing lesion.

Some authors[3,11] have pointed out that surgical and consequently pathological confirmation may be absent due to the surgeon's failure to open and examine the proximal colon. In a recent review of the subject, Schwartz et al[7] pointed out that at the time of surgery the colonic appearance and vascular pulsations may fail to indicate early abnormalities, if indeed such is secondary to ischemia. Perhaps this is the reason more cases have not been reported. The radiologist can play an instrumental role in alerting the surgeon to the presence of ulcerative disease and possibly prevent complications of healing at the surgical anastomosis.[11]

Our case, although shedding no light on the ultimate etiology, reinforces previous reports that indicate that partial obstruction of any cause may lead to proximal colitis.

Summary

A case is reported of ulcerative disease of the colon proximal to a benign intraluminal neoplasm. Barium enema showed diffuse generalized mucosal ulceration proximal to an intraluminal polypoid lesion pathologically proven to be a lipoma. A review of the possible etiologic factors of such ulcerative disease is discussed. To

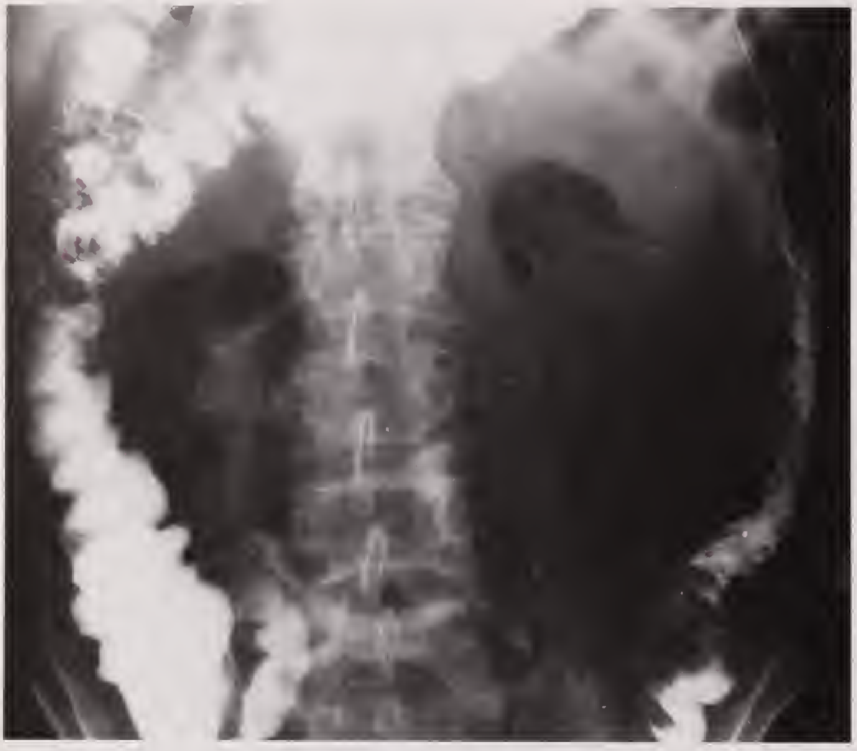

FIG. 3. Post-evacuation study. Generalized mucosal ulceration with loss of the normal post-evacuation mucosal pattern is demonstrated and loss of normal haustration.

date this is the first reported case occurring proximal to a benign colonic neoplasm.

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Adenoid Cystic Carcinoma of the Cervix with Myxochondroid Change*

Report of a Case

JOSEPH KOVI, M.D.†

Adenoid cystic carcinoma of the cervix is a very rare entity, only 22 cases having been reported.[1-6] The following is a report of an adenoid cystic carcinoma of the cervix with myxochondroid transformation in the stroma. A similar case has not been found in the literature.

Report of Case

A 31-year-old black woman, gravida VII, para VI, ab I, was admitted to the Department of Obstetrics and Gynecology of Howard University Medical College on September 8, 1971. She complained of "heavy" vaginal bleeding and abdominal pain. The cervix appeared enlarged and disfigured. On September 9, 1971 a cervical smear was obtained, and it was reported as positive for malignant cells. On September 15, 1971 a cervical biopsy was performed. The pathologic report was invasive anaplastic carcinoma of the cervix. A radical hysterectomy with node dissection and bilateral salpingoöphorectomy were done for invasive carcinoma of cervix state 2a on September 27, 1971 by Dr. Charles M. Cabaniss.

Pathologic findings. Submitted for pathologic study were a uterus with cervix, vaginal cuff, both fallopian tubes and ovaries, and regional lymph nodes (S71-3944). The uterus measured 9x6x3 cm. The external cervical os was partially occluded by tumor. On cut surface the entire cervix appeared to be replaced by white-gray tumor tissue.

Microscopic examination. Hematoxylin and eosin-stained sections revealed that virtually the entire cervix had been infiltrated by tumor tissue. The tumor showed a remarkable diverse composition in different areas. The predominant pattern was that of a poorly differentiated glandular carcinoma with vague suggestions of a cribriform pattern. In other areas the small, undifferentiated tumor cells with regular, hyperchromatic nuclei grew in cords and bands. Here the tumor cells formed various-sized cystic areas, containing faint, homogenous secretion. This so-called cribriform pattern is the characteristic histomorphologic feature of adenoid cystic carcinoma. Associated with these areas there were somewhat more differentiated glandular structures lined by cuboidal epithelium (Fig. 1). Not too far from the endocervix there were groups of ostensibly well-differentiated ducts, forming lobular structures, not unlike that found in the breast. These ductal formations seemed to be compressed by the huge amount of a cellular, faint-blue mucinous material surrounding them. Adjacent to the endo-cervical epithelium a few glands appeared to be lined partly with normal mucus-secreting cells, and partly by a non-mucus-secreting, rather atypical epithelium. Nearby there were numerous islands of myxochondroid. These were sharply delineated and stained deeply with hematoxylin. In this pseudo-cartilagenous

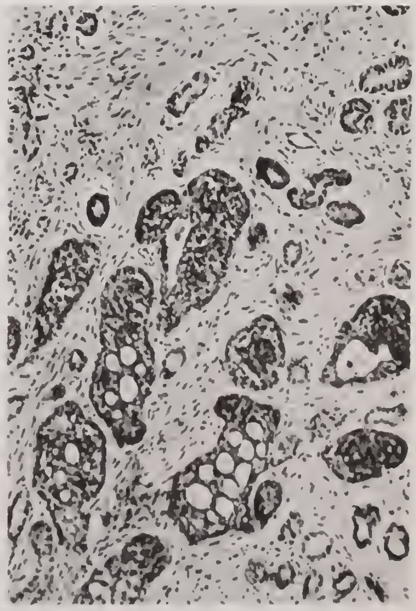

FIG. 1. Adenoid cystic carcinoma of the cervix with characteristic cribriform pattern showing a few better differentiated ducts.

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Reprint requests may be addressed to Dr. Kovi at the Department of Pathology, Howard University College of Medicine, 520 W Street, N.W., Washington, D.C. 20001 material the cells assumed a round form and were disposed singly or in small groups and the formation of capsules around the cells made them indistinguishable from chondrocytes. Nevertheless, the cells forming this pseudo-cartilagenous material showed all of the characteristics of malignancy, including a considerable number of mitotic figures (Fig. 2).

Tumor cells infiltrated in a fairly broad field the nearby subendometrial myometrium.

All of the 13 examined lymph nodes were negative for metastatic tumor cells.

Histochemical study of tumor. The faint, blue homogenous material in vacuoles of the cribriform areas surrounding the ducts, of the myxochondroid stained poorly with mucicarmin and gave a weak reaction with periodic acid-Schiff, both before and after diastase digestion. It stained heavily with alcian blue and gave a vigorous gamma metachromatic reaction with toluidine blue. After a three-hour incubation with hyaluronidase this gamma metachromasia diminished except in the pseudo-cartilage. Secretion of ducts lined by a cuboidal epithelium stained in contrast moderately with mucicarmin and reacted well with the PAS reagent both before and after diastase digestion. It stained poorly with alcian blue and gave a negative gamma metachromasia.

Discussion

From the reported few cases of cervical adenoid cystic carcinoma it appears that the disease affects predominantly elderly women.[1-6] In the present case, in which the tumor was associated with myxochondroid transformation in the stroma, the neoplasm developed in a fairly young patient.

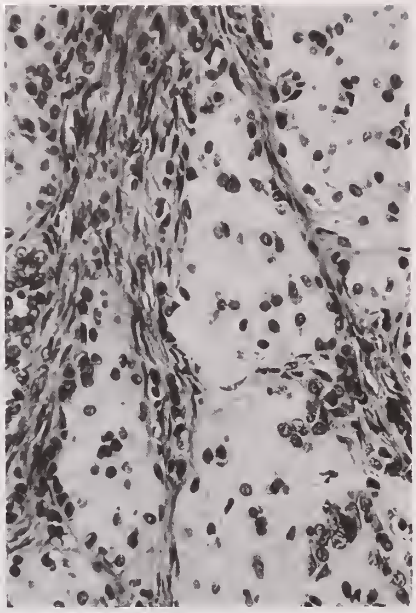

FIG. 2. Myxochondroid of the cervix showing atypical cells enclosed in capsules and resembling chondrocytes. One cell is in division.

Azzopardi and Smith[7] demonstrated that mucins in adenoid cystic carcinoma and mixed tumor of the salivary glands can be divided into two types: epithelial mucin, secreted by the true ducts, giving a strong PAS reaction both before and after diastase digestion, and negative gamma metachromasia, stromal mucin, exhibiting a poor PAS reaction but vigorous gamma metachromasia. Myxochondroid areas of the tumor in this case gave the histochemical reactions of stromal mucin.

It appears that the tumor arose from the endocervical mucous glands.

Summary

This is a report of an adenoid cystic carcinoma of the cervix associated with myxochondroid change in the stroma in a 31-year-old Negro woman. No case of this entity has yet been reported.

Acknowledgment

The technical assistance of Mrs. Kathryn F. Berry is gratefully acknowledged.

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Cardiac Tamponade and Constrictive Pericarditis*†

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Diagnosis of Cardiac Tamponade

The intrapericardial pressure in normal individuals is subatmospheric. Cardiac tamponade exists when the intrapericardial pressure is appreciably increased by fluid. Its severity depends upon the rate of accumulation and the volume of pericardial fluid. Important causes include trauma, infection, ruptured aortic aneurysm, and myocardial infarction

Equal compression of all cardiac chambers by the fluid explains the clinical picture: elevated venous pressure, pulmonary congestion, hypotension, and a quiet heart. One or 200 ml of fluid accumulated rapidly suffice to cause tamponade, as in trauma. In infectious and malignant effusions the onset, although less acute, may still be rapid. If an effusion collects over many days or weeks, several hundred ml may gather before tamponade ensues.

In extreme cases examination discloses impaired cerebral function, pallor, sweating, and impalpable or weak radial pulses. Cerebral symptoms are less prominent with milder tamponade, but the patient may note chest discomfort and dyspnea.

Pulsus paradoxus, an abnormal inspiratory fall in arterial pressure, is almost invariable with tamponade. The pulse diminishes or entirely disappears during each inspiration. Pulsus paradoxus may be difficult to elicit under three circumstances: profound hypotension, mild tamponade, and tachypnea. When the radial pulse is impalpable, paradoxus may be elicited from the femoral pulse. In order to appreciate pulsus paradoxus in mild cases it may be necessary to have the patient breathe as deeply and slowly as possible without straining at the end of inspiration or expiration. However, when the aim is to measure the severity rather than simply detect pulsus paradoxus, respiration should be maintained as normal as possible because hyperventilation exaggerates pulsus paradoxus. Blood pressure is measured with a cuff, releasing the pressure as slowly and evenly as possible. Initially, systolic sounds are audible only during expiration, but as cuff pressure falls, a point is reached when the sounds are heard throughout the respiratory cycle. The pressure difference between these two points estimates pulsus paradoxus.

Elevated venous pressure is best observed with the thorax elevated, frequently to 90 degrees. Venous pulsations are visible inferior and anterior to the ear and display a single slow expansion followed by a rapid collapse synchronous with the carotid pulse. Normal inspiratory variation and fall in venous pressure are often maintained. Precordial activity is absent or diminished, but if the apex beat is palpable cardiac dullness may be percussed lateral to it. The heart sounds are usually normal, sometimes muffled. A pericardial friction rub may be heard and does not preclude effusion. The ECG may show sinus tachycardia, decreased voltage, elevated ST segments, and in severe cases electrical alternans. The chest roentgenogram often reveals a globular increase in the cardiopericardial silhouette with disproportionately clear lung fields. If conditions permit fluoroscopy, it will be observed that the cardiac borders hardly pulsate while the aortic pulsations are vigorous. Hilar vessel shadows are often obliterated.

The presence of pericardial effusion can be confirmed most conveniently and safely by echocardiography. When this facility is not available special radiological procedures such as intravenous CO_2 injection or angiocardiography may be employed.

Diagnosis of Constrictive Pericarditis

In this disorder the heart is compressed not by fluid but by the visceral pericardium. In the majority of cases all cardiac chambers are equally involved, and this explains the hemodynamic similarities to tamponade. Intrapericardial pressure cannot vary during the respiratory and cardiac cycles because the pericardial space is obliterated. This explains the differences from tamponade. The etiology is unknown in most instances, tuberculosis being much less common than formerly. Other important causes include malignancy, therapeutic radiation, collagen diseases, and infections.

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Chronic constrictive pericarditis develops insidiously over many years. The pericardium often becomes heavily calcified. In recent series many cases are subacute and develop in weeks or months, a difference which reflects the changing etiology.

Patients with chronic constrictive pericarditis frequently are cachectic, pale, mildly jaundiced, and cyanosed. The venous pressure is greatly elevated, and the arterial pulse pressure may be small, although pulsus paradoxus occurs in only a third of the cases. The venous pulse displays two rapidly collapsing waves, one in systole, the other in diastole. The marked diastolic collapse may help to differentiate this condition from tamponade. The positive filling waves are much less prominent. The normal decrease in venous pressure with inspiration is absent, and paradoxical expansion may be seen.

Elevated venous pressure, hepatic and renal underperfusion and congestion, and protein-losing enteropathy combine to make edema, often with disproportionate ascites, a prominent feature of the disease. The liver may be enlarged or cirrhotic and its function severely impaired.

Precordial activity is absent or decreased. Sometimes systolic retractions are found. A loud protodiastolic third heart sound is heard all over the precordium. The ECG may show wide bifid P waves until atrial fibrillation supervenes. QRS voltage is decreased, and the T-wave is inverted in most leads. The heart appears normal in size to moderately enlarged on chest roentgenograms. Calcification may be seen readily on plain chest films, especially in the lateral projection. Thus the radiologist may be the first to call attention to the correct diagnosis. Fluoroscopy confirms calcification and reveals decreased amplitude of motion of the heart walls.

Cachexia, anasarca, liver failure, and a high venous pressure constitute a striking clinical picture. Nevertheless, the diagnosis is often missed because the patient is considered to have heart failure or primary liver disease. However, the venous pressure is normal in non-cardiopericardiac causes of edema. If the pericardium is not calcified, final distinction from myocardial disease may require cardiac catheterization.

Subacute cases show less cachexia and liver damage. One can often obtain a history of recent acute pericarditis or chest trauma. The pericardium is not calcified, and pulsus paradoxus is more common than in the chronic form. The diagnosis should be suspected in any patient with unexplained weight gain, dyspnea, edema, hepatomegaly, and raised venous pressure who does not have heart disease of easily recognized etiology.

Treatment of Cardiac Tamponade

The only treatment is removal of the fluid. If the patient is moribund, pericardiocentesis is carried out as an emergency. More often, the patient should be moved to the operating suite, intensive care area, or cardiac catheterization laboratory. Of the several approaches to the pericardium, the xiphisternal is the safest. The patient rests supine with the thorax elevated 45 degrees. The skin and subcutaneous tissues a few mm from the xiphisternum are cleansed and infiltrated and then pierced with the tip of a number 11 blade. An 18T needle is advanced posteriorly until its tip has negotiated the boney margin of the thorax. The needle is then advanced close to the midline with a slight posterior tilt until the pericardium is reached. If there is much effusion little or no cardiac pulsation will be felt. The needle is advanced through the pericardium, usually with a palpable sensation.

A conventional ECG lead should be monitored throughout the procedure. If it is absolutely certain that all equipment is properly grounded, the position of the needle tip can be monitored by a direct ECG lead. A cable connects the needle hub and an indifferent electrode (e.g., a limb lead) to the electrocardiograph which is set in position "V." If the needle tip touches the epicardium a large ST-segment elevation occurs in the needle lead and a smaller one in the conventional lead. Also, ventricular extrasystoles may be provoked. When these phenomena occur the needle should not be advanced further but withdrawn slowly and carefully until the ST-segments are again isoelectric.

As soon as the pericardium is entered, a sample is aspirated, and if bloody its hematocrit and clotting time are compared with those of the patient's blood. Hemorrhagic fluid rarely clots and almost always has a lower hematocrit than venous blood. Pericardial pressure should then be measured with a manometer, after which samples should be obtained for bacteriology, chemistry, and cytology. Removal of the first 50-200 ml of fluid usually produces prompt relief because of the steep pericardial pressure volume curve. Venous pressure and pulsus paradoxus decrease and the arterial pulses become full. Pericardial fluid is aspirated as long as it comes off easily, and preferably until the pericardial pressure is zero or less. No elaborate precautions to prevent air aspiration are needed; indeed, introduction of a volume of air or CO_2 equal to less than half the aspirated fluid volume is useful to delineate the pericardium in subsequent chest roentgenograms.

Sometimes it is desirable to leave a small tube in the pericardium. Chemotherapeutic agents may be instilled into the pericardial sac. A chest roentgenogram is obtained at the end of the procedure. Appropriate systemic antibiotic therapy is commenced, and, if tuberculosis is a reasonable possibility, steroids may be administered as well.

If a delay in initiating pericardiocentesis is inevitable, the circulation can be temporarily sustained by infusion of isoproterenol together with volume expansion by means of dextran blood or saline. Isoproterenol is started at 2-4 micrograms per minute and increased until perceptible effects on the pulse and pressure are evident. Recurrence of tamponade may require repetition of pericardiocentesis, but surgical drainage may then be preferable. In traumatic cases recurrence after one or two successful aspirations is an indication for surgical drainage. When tissue diagnosis is important and a physician experienced in the technique of pericardiocentesis is not available, the pericardium can simply be drained and biopsy specimen taken under local anesthesia after excision of the xiphoid process.

Treatment of Constrictive Pericarditis

Once this diagnosis is established the patient should be referred to a thoracic surgeon for pericardiectomy. Patients improve following digitalization because of the frequently associated myocardial involvement. Tachycardia secondary to atrial fibrillation responds well to digitalis, as in heart failure. These measures alone produce diuresis, but it is usually advisable to administer diuretics as well. These are given in high dosage for as long as they are effective and serum sodium and chloride levels are not excessively depressed. Serum potassium must be maintained at normal levels by means of oral potassium chloride administration, and salt intake should be moderately reduced. Because of liver congestion, alcohol and other hepatic toxins must be forbidden. These measures may be employed to effect an immediate improvement and to prepare the patient for pericardiectomy. They are also remarkably successful for variable periods in patients who are unwilling to undergo operation, but in these circumstances slow progression of physiologic decompensation invariably occurs.

Pericardiectomy is now a safe operation. In a young individual without pericardial calcification, cardiac enlargement, or atrial fibrillation the mortality is virtually zero. Thus the operation is urgently indicated in subacute constrictive pericarditis. In chronic calcific constrictive pericarditis the operative risk is 5-10% in the hands of experienced cardiac surgeons. Pericardiectomy is not an open-heart procedure, but full cardiopulmonary bypass facilities should be immediately available to deal with cardiac trauma, evidence of which may appear suddenly at any time during the operation.

Several months elapse after operation before recovery is complete, especially in the more chronic cases. Venous pressure is reduced somewhat immediately following the operation but only slowly returns to normal. Digitalis and diuretics may be required for several months to control edema and effusions. In the most chronic cases with heavy calcification and long-standing congestion the aim is improvement rather than cure. The results in these patients are limited by myocardial involvement, hepatic insufficiency, chronic pulmonary congestion, and pleural disease.

Cardiac compression secondary to malignant disease should be managed as conservatively as possible. Pericardiocentesis and the instillation of cancer chemotherapeutic agents may control the process in some patients. In others it may be necessary to construct a pericardiopleural window to drain the pericardium via the pleural space. There is little to be done for patients with constrictive pericarditis secondary to malignancy without an element of pericardial effusion. Surgeons are generally reluctant to perform pericardiectomy under such circumstances.

Radionuclide Angiocardiography*†

JOEL S. KARLINER, M.D.

Standard methods for visualization of the heart's chambers and adjacent great vessels require cardiac catheterization followed by the rapid injection of radiopaque contrast material into the circulation under high pressure. The procedure is not without hazard or discomfort to the patient. Furthermore, although serial studies are possible, repeated catheterizations of most individuals are potentially hazardous. Injection of radionuclides, however, can be accomplished rapidly from a peripheral site without discomfort to the patient. The procedure entails no significant risk or hemodynamic alterations. During the past few years a number of studies have indicated that radionuclide angiocardiography can be used for the qualitative as well as the quantitative assessment of the cardiovascular system.

Qualitative Studies

Peripheral intravenous injection of a bolus of 99m technetium pertechnetate permits sequential visualization of the heart and great vessels. The radionuclide has a half-life of four to six hours. The radiation exposure (0.06-0.16 rads/dose) is in the range of conventional diagnostic X-rays. The image is recorded on an Anger scintillation camera, stored, replayed, and photographed. This technique has been used in the diagnosis of a variety of congenital and acquired cardiac diseases, including left-to-right shunts, transpositon complexes, coarctation of the aorta, mitral and aortic valve disease, ventricular aneurysm, and left atrial myxoma. In addition, the sequential appearance of the radionuclide bolus may be inscribed in a fashion similar to an indicator dilution curve. From mathematical analysis of the curve the presence or absence of a left-to-right shunt can be determined. The rapidity and safety of the procedure, which does not require hospitalization or special preparation of the patient, favors its use as a diagnostic screening test, especially in children with indeterminate murmurs in whom congenital heart disease is suspected.

Recent studies indicate that it is possible to obtain accurate qualitative information relative to the regional vascular supply of the heart in patients with coronary artery disease. Macroaggregated albumin particles labeled with ^{131}iodine can be injected into one coronary artery, and similar particles labeled with 99m technetium can be injected into the other coronary vessel. No changes in the heart rate, ECG, or systemic blood pressure occur during or following injection of the radioactive material. Electronic pulse-height discrimination allows gross assessment of perfusion by each coronary artery. Although this technique is investigational at the present time, it may find application in the selection and follow-up of patients undergoing myocardial re-vascularization procedures.

Quantitative Studies

It would be desirable to have a reliable method for quantitative assessment of left ventricular performance in patients who are acutely ill. Furthermore, a simple, virtually noninvasive technique would permit serial studies without subjecting patients to repeated cardiac catheterizations. In earlier experimental studies in dogs it was demonstrated that ventricular volumes could be quantitated by isotope visualization of the heart. These studies have now been extended to patients. Left ventricular volume measurements obtained by standard angiocardiographic techniques have been shown to correlate well with volumes calculated by isotope methods.

Another useful cineangiocardiographic method for quantitating left ventricular function has been the estimation of the ejection fraction. The stroke volume is the difference between the end-diastolic and the end-systolic volumes. The ratio of the stroke volume to the end-diastolic volume is termed the ejection fraction. The average normal systolic ejection fraction in man is 67%. Recently it has been shown in our laboratory as well as by others that accurate determinations of the ejection fraction can be obtained in patients by radionuclide techniques.

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* Submitted to the MEDICAL ANNALS by the Washington Heart Association.

† From the Cardiovascular Division, Department of Medicine, University of California at San Diego.

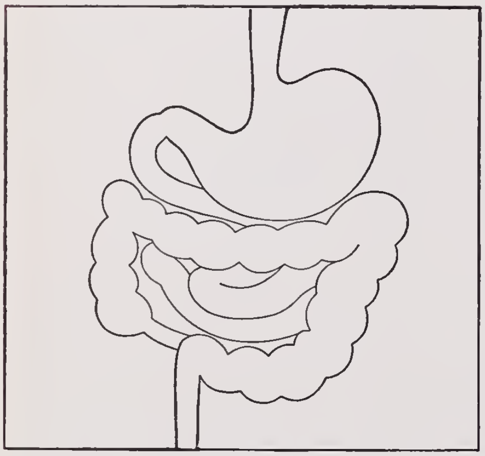
Reprint requests may be addressed to Dr. Karliner at the Cardiovascular Division, Department of Medicine, University of California at San Diego, P.O. Box 109, La Jolla, California 92037.

As indicated earlier, two advantages of the radioisotope method are that it does not disturb cardiac function and can be performed following a peripheral bolus injection through a very small-bore needle. In practice the radioisotope angiocardiogram is obtained with an Anger-type scintillation counter after the injection of 7-15 mc of 99m technetium as sodium pertechnetate. The sequentially obtained image is recorded and stored on magnetic tape along with the simultaneously recorded electrocardiogram. Both the stored video signal and the electrocardiogram are then replayed. By means of a gating discriminator triggered by the electrocardiogram, images at either end-diastole or end-systole are displayed on an oscilloscope. The cardiac silhouettes at each of these selected times are photographed directly, projected on a screen, and traced. Their areas are then determined and volumes and ejection fraction calculated.

Using this technique we have been able to document reduced left ventricular performance in patients acutely ill with myocardial infarction. It is also possible to diagnose abnormalities of left ventricular wall-motion with this method. In the future it will be possible to analyze serial changes in left ventricular function and wall-motion in acutely ill patients as well as in those who are recovering from myocardial infarction and myocardial revascularization procedures.

Nuclear medicine has made considerable contributions to the study of other organ systems. Its potential usefulness in the study of the heart and circulation has now been recognized, and future investigations may be expected to yield even more information.

in "Gasspastic" conditions



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The Physician as Witness
The Problem of Dying Declarations

When a physician is called upon to serve as a witness for a criminal or civil case, he encounters one of the most problematic and confounding aspects of law—the field of evidence. The technicalities surrounding the admissibility of certain testimony can be baffling to the lawyer and layman alike. One of the areas that is particularly notorious for its peculiarities is that of "dying declarations." A situation in which a doctor can be involved with this problem area is illustrated by the following hypothetical example:

Suppose a man suffering from multiple gunshot wounds is brought to the emergency room of the hospital with which you are affiliated. As attending physician you examine his condition and initiate emergency treatment. During these procedures the patient gasps out what he feels to be the identity of his assailant and the alleged motivations of the attack. Shortly thereafter the patient dies, due to his massive wounds. You relate to the police your conversation with the deceased; based upon your statements and further independent investigation, the police decide to arrest the man referred to by the decedent. At the subsequent trial, if you are called upon to testify, will you be allowed to relate what the patient told you immediately prior to his death? The answer is not obvious and depends upon several factors.

The basic objection against the admissibility of your testimony concerning the declarations of the decedent would be that such statements are "hearsay." Hearsay is a legal term and has often been defined, in legalese, as an "extra-judicial declaration used to prove the truth of the matter asserted therein." In essence, the statements made by the deceased in the hypothetical are hearsay because they were spoken outside of the present courtroom [extra-judicial] and are now being presented to prove the guilt of the present defendant [the matter asserted therein].

Statements characterized as hearsay are not allowed into evidence unless they fall into one of the recognized exceptions to the hearsay rule. In the present situation, the relevant exception is the "dying declaration" doctrine. Basically, the "dying declaration" exception covers statements made by a person in the face of his own death. Many courts have so severely limited the scope of the "dying declaration" doctrine as to render it useless in most situations.

The courts in Maryland, Virginia and the District of Columbia uniformly take a conservative view of the doctrine. In general, they have set forth five prerequisites which must be met before a physician will be allowed to testify during a trial as to what a deceased patient told him.

The first condition is that the patient must have been conscious that death was near and certain at the time that he made his statements. The purpose for this requirement involves the basic justification given for the entire doctrine. Most exceptions to the hearsay rule are based on the presence of the two factors of (1) necessity and (2) trustworthiness. In this case, the necessity of the doctor's testimony is obvious as the original declarant is dead. The trustworthiness of the original statement is not so obvious. The courts have been willing to assume that the declarations of a dying man are trustworthy since "no one wishes to meet his maker with a lie upon his lips." Although this presumption is psychologically impossible to prove, it remains as a basis for the doctrine and for the qualification that the patient realize the gravity of his situation.

The actual content of the statements of the declarant can be crucial in establishing the first condition. For example, if the patient stated that he longed for a speedy recovery so that he could wreak vengeance upon his attacker, the result would be different than if he had given you frantic instructions on the future care of his family. Another important consideration would be any statements which the physician himself made to the patient concerning the patient's condition. If the patient had been advised of the seriousness of his condition, the result would be different than if you had attempted to reassure him. Finally, the apparent fatality of the wounds can of itself be sufficient to raise the presumption that the declarant realized that his death was imminent. It is interesting to note that the declarant need not die immediately after the statement was made so long as death was certain at the time of the statement, death was attributable to the wounds inflicted by the attacker, and death occurred prior to trial.

In fact, the second prerequisite is that the declarant must actually be dead at the time of the trial. The reason for this is obvious; if the patient were still alive, there would be no necessity for the physician's testimony.

The third prerequisite to admissibility is perhaps the most controversial. In the local jurisdictions, statements made by the deceased are admissible only in criminal prosecutions for homicide. Consequently, if your patient was a victim of a brutal rape and subsequently dies of wounds inflicted in the attack, and if the accused was later prosecuted for rape but not for homicide, your testimony would be inadmissible. Furthermore, since the admissibility is limited to criminal prosecutions for homicide, your testimony may be precluded in the subsequent civil wrongful death action in which the family of the victim would sue the defendant for monetary damages.

The fourth factor allows the declarations to be admissible only insofar as they relate to the circumstances of the killing and the events immediately surrounding it. This factor is fairly vague; however, it stands for the proposition that statements which describe a long-standing animosity between the deceased declarant and his alleged assailant would be inadmissible, whereas statements describing the actual attack itself would be admissible.

The final factor requires the declarant to have had an adequate opportunity to observe the events which he described. If the situation is such that it is apparent that the patient had no opportunity to see his assailant, then his opinions as to the probable identity of the assailant may not be recounted at a subsequent trial.

Although the "dying declarations" doctrine has been criticized for being overly restrictive, it has remained intact in its conservative form, as above limited, in the local jurisdictions. By considering the many prerequisites which must be met, it should be evident that even the most revealing and accusatory statements made by your patient need not necessarily have any subsequent legal significance.

WILLIAM CHARYK

Edited by Stefan F. Tucker of Arent, Fox, Kintner, Plotkin and Kahn, Washington, D.C.

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The first clinic for the diagnosis and treatment of children with handicapping conditions was created in New York City during the middle of the last century. Shortly thereafter, the Cleveland Rehabilitation Center was started to meet the needs of children with crippling conditions. At the turn of the century, the National Society for Crippled Children was organized, followed by the National Foundation for Infantile Paralysis.

Federal funds were made available to States through the Social Security Act of 1935 "to extend and improve services for locating crippled children and for providing medical, surgical, corrective, and other services and care." These grants-in-aid originally were administered by the Children's Bureau, and in 1969, the Administrator of the Health Services and Mental Health Administration, HEW, assumed this responsibility.

Almost a half a million handicapped youngsters were served by State crippled children's agencies by the end of the last decade.

Money grants have been provided to the District of Columbia since 1938 on a matching basis. These funds were used to start the Crippled Children's program and services and facilities were expanded through the years.

The services of the Crippled Children's Division include:

1. Crippled Children's Unit on the grounds of D.C. General Hospital—in-patient and out-patient care is available. Clinics include: Pediatric, Orthopedic, Cardiac, Eye, Audiology and Speech, Neurology, Convulsive Disorder, Cleft Palate, Amputee, Rehabilitation, Dental and Genitourinary. Social Services, Occupational Therapy, Physical Therapy, Psychological Services and Prosthetics also are provided.
2. Gales Clinic, 65 Massachusetts Avenue, N.W.—Cardiac Screening, Eye Clinic, Audiology Clinic, Allergy Clinic, Clinic for Retarded Children, Preschool Diagnostic Clinic, and School Diagnostic Clinic.
3. Melvin Sharpe Health School, an educational facility for children with handicapping conditions, is located at 4300 13th Street, N.W. Occupational and physical therapists are assigned from the Crippled Children's Division to provide services to those youngsters who need therapy.
4. Hearing screening is offered by the Division's technicians or school nurses. Children in first, third, seventh, and tenth grades are screened annually, as are selected referrals. Some 40,000 children receive these screening tests in schools each year. About 5 percent fail the test and are referred to Gales Clinic for another test. Those youngsters with hearing defects are cared for by medical specialists at the Crippled Children's Unit.

One of the unique programs at the Crippled Children's Unit is the Juvenile Amputee Program. This Program is part of the National Academy of Sciences' management of young amputees project, and is one of some two dozen regional clinics in the United States. Admitted to the Juvenile Amputee Program are residents from Maryland and Virginia, as well as the District of Columbia. More than 100 youngsters are unilateral or quadruple amputees. The medical conditions may be the result of a congenital defect, accident, or surgery.

Eligible to take part in the Crippled Children's program are those under age 21 who have or are suspected of having any of these handicaps: orthopedic and neuro-muscular, including amputations; cardiac, including rheumatic fever and rheumatic and congential heart diseases; visual defects; communication disorders, including hearing, speech, and language pathology; neurological handicaps, including convulsive disorders; cleft lip and palate and other congenital anomalies; defects requiring plastic surgery; cerebral palsy and other multiple handicaps; mental retardation; allergies; and other long term handicapping conditions. In-patient hospital services are available only to eligible District residents. Eligibility is construed to mean holders of Medicaid or D.C. medical eligibility cards.

Referrals to the Crippled Children's program come from Department of Human Resources facilities or private physicians. Some of the patients are self referrals. Available to patients are specially designed corrective shoes, braces, prosthesis, hearing aids, and other mechanical devices.

Program staff coordinate medical activities with other agencies such as the Board of Education, other governmental agencies, and non-official agencies.

Dr. Merle McPherson is in charge of more than 100 program staff and approximately one million dollar budget. Physicians may call Dr. McPherson at 629-3058 for information about admitting procedures, or you may call the Crippled Children's Unit on the grounds of D.C. General Hospital at 626-5214, or Gales Clinic at 629-4773.

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The Department of Radiology
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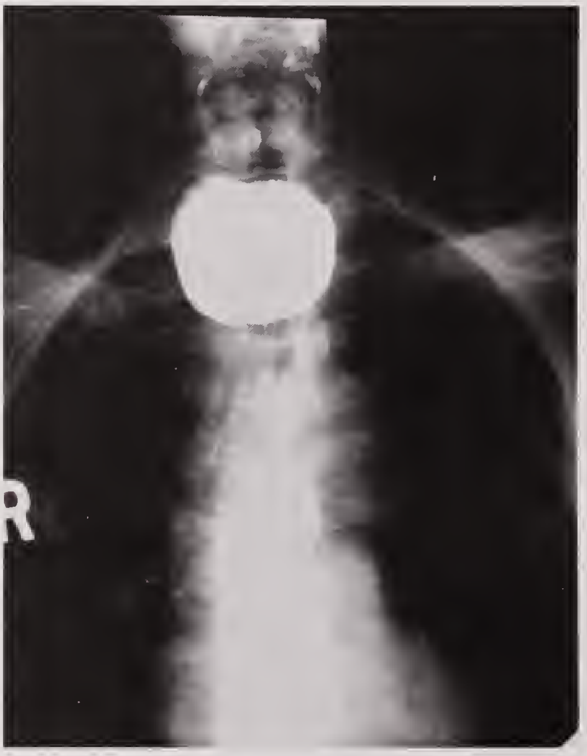

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(For diagnosis see page 669)

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Glyceryl guaiacolate	50.0 mg.
Dextromethorphan hydrobromide	7.5 mg.

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Select the Robitussin® "Clear-Tract" Formulation That Treats Your Patient's Individual Coughing Needs:

All 5 Robitussins have an EXPECTORANT-DEMULCENT action. Keep this handy chart as a guide in selecting the formula that provides the *extra* benefits you want for your patient.

Robitussin® extra benefit chart	Cough Suppressant	Antihistamine	Long-Acting (6-8 hours)	Nasal, Sinus Decongestant	Non-Narcotic
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ROBITUSSIN A-C®	●	●			
ROBITUSSIN-DM®	●		●		●
ROBITUSSIN-PE®				●	●
COUGH CALMERS®	■		■		■

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Richmond, Virginia 23220

A-H-ROBINS

Everybody experiences psychic tension.

Most people can handle this tension.

Some people develop excessive psychic tension and need your counseli

and a few may need counseling
and the psychotropic action of Valium® (diazepam).

Before deciding to make Valium
(diazepam) part of your treatment
lan, check on whether or not the
atient is presently taking drugs
nd, if so, what his response has
een. Along with the medical and
ocial history, this information can
elp you determine initial dosage,
he possibility of side effects and
he ultimate prospects of success
r failure.

While Valium can be a most
elpful adjunct to your counseling,
t should be prescribed only as long
s excessive psychic tension per-
ists and should be discontinued
hen you decide it has accom-
lished its therapeutic task. In
eneral, when dosage guidelines
re followed, Valium is well
olerated (see Dosage). For con-
enience it is available in 2-mg, 5-mg
nd 10-mg tablets.

Drowsiness, fatigue and ataxia
ave been the most commonly re-
orted side effects.

Until response is determined,
atients receiving Valium should
e cautioned against engaging in
azardous occupations requiring
omplete mental alertness, such
s driving or operating machinery.

ROCHE
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Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders, athetosis, stiff-man syndrome, convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence. In pregnancy, lactation or women of childbearing age, weigh potential benefit against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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(diazepam)

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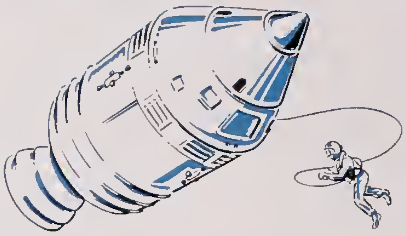

Man in space, now fait accompli, re-emphasizes the importance of Uro-Phosphate therapy. Research into the effect of space travel on the astronaut reveals that weightlessness causes loss of bone calcium. As the bones are required to bear less and less of the weight of the body they lose calcium, increasing the calcium content of the urine. When physical activity is reduced, the acidity of the urine should be adjusted to keep increased calcium in solution . . . . a prophylaxis to prevent kidney or bladder calculi.

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Each tablet contains: METHENAMINE, 300 mg.; SODIUM ACID PHOSPHATE, 500 mg.

Uro-Phosphate gives comfort and protection when inactivity causes discomfort in the urinary function. It keeps calcium in solution, preventing calculi; it maintains clear, acid, sterile urine; it encourages complete voiding and lessens frequency when residual urine is present.

Dosage:

For protection of the inactive patient 1 or 2 tablets every 4 to 6 hours is usually sufficient to keep the urine clear, acid and sterile.

2 tablets on retiring will keep residual urine acid and sterile, contributing to comfort and rest.

A clinical supply will be sent to physicians and hospitals on request.

Uro-Phosphate contains sodium acid phosphate, a natural urinary acidifier. This component is fortified with methenamine which is inert until it reaches the acid urinary bladder. In this environment it releases a mild antiseptic keeping the urine sterile.

Uro-Phosphate is safe for continuous use. There are no contra-indications other than acidosis. It can be given in sufficient amount to keep the urine clear, acid and sterile. A heavy sugar coating protects its potency.

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with Intrinsic Factor

(See reverse side for prescribing information.)

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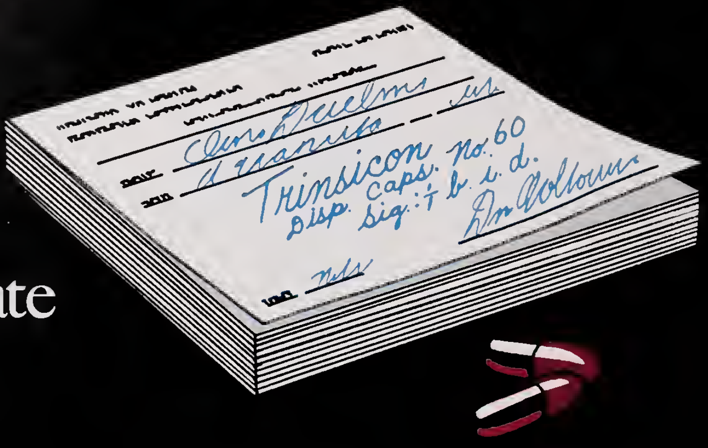
Trinsicon no. 60
Disp. caps. Sig: i b.i.d.

Description: Each Pulvule® contains—

Special Liver-Stomach Concentrate, Lilly (containing Intrinsic Factor)	240 mg.
Cobalamin Concentrate, N.F., equivalent to Cobalamin	7.5 mcg.
Iron, Elemental (as Ferrous Fumarate)	110 mg.
Ascorbic Acid (Vitamin C)	75 mg.
Folic Acid	0.5 mg.

(The total vitamin B_{12} activity in the Special Liver-Stomach Concentrate, Lilly, and the Cobalamin Concentrate, N.F., is 15 micrograms.)

Indications: Trinsicon is a multifactor preparation effective in the treatment of anemias that respond to oral hematinics, including pernicious anemia and other megaloblastic anemias and also iron-deficiency anemia. Therapeutic quantities of hematopoietic factors that are known to be important are present in the recommended daily dose.

Vitamin B_{12} with Intrinsic Factor—When secretion of intrinsic factor in gastric juice is inadequate or absent (e.g., in Addisonian pernicious anemia or after gastrectomy), vitamin B_{12} in physiological doses is absorbed poorly, if at all. The resulting deficiency of vitamin B_{12} leads to the clinical manifestations of pernicious anemia. Similar megaloblastic anemias may develop in fish tapeworm (*Diphyllobothrium latum*) infection or after a surgically created small-bowel blind loop; in these situations, treatment requires freeing the host of the parasites or bacteria which appear to compete for the available vitamin B_{12}. Strict vegetarianism and malabsorption syndromes may also lead to vitamin B_{12} deficiency. In the latter case, parenteral therapy, or oral therapy with so-called massive doses of vitamin B_{12}, may be necessary for adequate treatment of the patient.

Potency of intrinsic factor concentrates is determined physiologically, i.e., by their use in patients with pernicious anemia. The liver-stomach concentrate with intrinsic factor and the vitamin B_{12} contained in two Pulvules Trinsicon provide 1½ times the minimum amount of therapeutic agent which, when given daily in an uncomplicated case of pernicious anemia, will produce a satisfactory reticulocyte response and relief of anemia and symptoms.

Concentrates of intrinsic factor derived from hog gastric, pyloric, and duodenal mucosa have been used successfully in patients who lack intrinsic factor. For example, Fouts *et al.* maintained patients with pernicious anemia in clinical remission with oral therapy (liver extracts or intrinsic factor concentrate with vitamin B_{12}) for as long as twenty-nine years.

After total gastrectomy, Ficarra found multifactor preparations taken orally to be "just as effective in maintaining blood levels as any medication that has to be administered parenterally." His study was based on twenty-four patients who had survived for five years after total gastrectomy for cancer and who had been taking two Pulvules Trinsicon daily.

Folic Acid—Folic acid deficiency is the immediate cause of most, if not all, cases of nutritional megaloblastic anemia and of the megaloblastic anemias of pregnancy and infancy; usually, it is also at least partially responsible for the megaloblastic anemias of malabsorption syndromes, e.g., tropical and nontropical sprue.

It is apparent that in vitamin B_{12} deficiency (e.g., pernicious anemia), lack of this vitamin results in impaired utilization of folic acid. There are other evidences of the close folic acid-vitamin B_{12} interrelationship: (1) B_{12} influences the storage, absorption, and utilization of folic acid, and (2), as a deficiency of B_{12} progresses, the requirement for folic acid increases. However, folic acid does not change the requirement for vitamin B_{12}.

Iron—A very common anemia is that due to iron deficiency. In most cases, the response to iron salts is prompt, safe, and predictable. Within limits, the response is quicker and more certain to large doses of iron than to small doses.

Each Pulvule Trinsicon furnishes 110 mg. of elemental iron (as ferrous fumarate) to provide a maximum response.

Ascorbic Acid—Vitamin C plays a role in anemia therapy. It augments the conversion of folic acid to its active form, folinic acid. In addition, ascorbic acid promotes the reduction of ferric iron in food to the more readily absorbed ferrous form. Severe and prolonged vitamin C deficiency is associated with an anemia which is usually hypochromic but occasionally megaloblastic in type.

Contraindications and Precautions: Anemia is a manifestation that requires appropriate investigation to determine its cause or causes.

Folic acid *alone* is unwarranted in the treatment of pure vitamin-B_{12}-deficiency states, such as pernicious anemia. Indeed, the use of folic acid in large doses in pernicious anemia without adequate vitamin B_{12} may result in hematologic remission but neurological progression.

As with all preparations containing intrinsic factor, resistance may develop in some cases of pernicious anemia to the potentiation of absorption of physiological doses of vitamin B_{12}. If resistance occurs, parenteral therapy, or oral therapy with so-called massive doses of vitamin B_{12}, may be necessary for adequate treatment of the patient. No single regimen fits all cases, and the status of the patient observed in follow-up is the final criterion for adequacy of therapy. Periodic clinical and laboratory studies are considered essential and are recommended.

In extremely rare instances, skin rash suggesting allergy has been noted following the oral administration of liver-stomach material. Allergic sensitization has been reported following both oral and parenteral administration of folic acid.

Hemochromatosis and hemosiderosis are contraindications to iron therapy.

Adverse Reactions: In rare instances, iron in therapeutic doses produces gastrointestinal reactions, such as diarrhea or constipation. Reducing the dose and administering it with meals will minimize these effects in the iron-sensitive patient.

Dosage: One Pulvule twice a day. (Two Pulvules daily produce a standard response in the average uncomplicated case of pernicious anemia.)

How Supplied: Pulvules Trinsicon® (hematinic concentrate with intrinsic factor, Lilly), in bottles of 60 and 500 and in Identi-Dose® (unit dose medication, Lilly) in boxes of 100.

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book REVIEWS

Natural History of Infectious Disease. J. A. BOYCOTT, M.A., D.M. Price, $3.95. Pp. 42, New York: St. Martin's Press, 1971.

This book is one of a series which covers the field of biology for high school students in Great Britain. It is not intended to be a Textbook of Medicine.

The purpose of the book is to give an overview of Infectious Disease as an "example of the phenomena of parasitism." In 42 pages the nature of infectious diseases, the host-parasite relationship, defense mechanisms and epidemiology are discussed. It is quite comprehensive in scope and avoids details which would blunt the thrust of the effort.

The book is easily read and is an excellent introduction to the concepts of infectious diseases. It is the kind of a book which might pique the curiosity of a high school student and be a stimulus to further reading in medical and biological sciences.

JAMES A. CURTIN, M.D.
Chairman, Department of Medicine
Washington Hospital Center

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"Service is our Specialty"

Water Treatment and Examination. W. S. HOLDEN, Editor. Price, $27.50. Pp. 513, with illustrations. Baltimore: Williams and Wilkins, 1970.

This book is the successor to eight previous editions of *The Examinations of Water and Water Supply* edited by Dr. E. Wendel Taylor and published for the Society for Water Treatment and Examination in England. The original editions have been updated and expanded commensurate with recent interest in research pertaining to the quantity and quality of water supply in our environment. The 43 chapters are complemented by several appendices and are well indexed. Chapters deal with basic water chemistry with regard to both organic and inorganic constitutants, water pollution and the various techniques to purify drinking water. Of particular interest to physicians and public health personnel are several chapters devoted to bacteriologic contamination of water and the methods for such assessment, analyses, and indices for purification. Separate chapters deal not only with the customary coliform group, which is the basis for measuring water purity, but also with proteus, pseudomonas, fecal streptococci, spore forming anaerobes, spirochetes and other pathogenic organisms. In addition, there is a rather extensive chapter devoted to viruses in drinking water which is very up-to-date and includes much recent epidemiologic data as well as available information on the effect of storage in reservoirs and current purification processes on viruses. In addition to many chapters dealing with the several phases of purification, other subjects covered include augmentation of supply by desalination and recycling and three chapters devoted to the consumer dealing with the purification of water for swimming pools, the color, odor and taste in water and a final chapter devoted to common complaints from consumers.

Portions of the text which I read were very concise, clear, illustrations were good and the material well documented with tables and each chapter is appended by a list of pertinent references. Although many of the tables detail findings in British waters, available data suggest the pollution problems found in this country are similar.

The book comprises a review of the literature through 1969 and is superbly done and I recommend it to public officials responsible for this problem, to epidemiologists and public health officials, and to physicians with an abiding interest in water borne disease and health implications of our drinking supply.

STUART H. DANOVITCH, M.D.
Chairman, Environmental Health Committee
Medical Society of D.C.

Living With Your Bad Back. THEODORE BERLAND and ROBERT G. ADDISON, M.D. Price, $5.95. Pp. 151. New York: St. Martin's Press, 1972.

Theodore Berland is a free-lance writer who has co-authored several books on medical subjects with physicians. Dr. Addison is an orthopedic surgeon who is very knowledgeable concerning the neuromusculoskeletal problems of the low back. This book was written for the lay public with low back problems. It is an easy reading 145 page book which succeeds in giving the person with back trouble a reasonable insight into the basis of his problem and how best to handle it. The flow of the book is rational, progressing from an acute attack, through the multiple aspects of care and on to a reasonable conclusion. It explains how a person who has had a bad back can live a satisfying and pain-free existence in the presence of his disability. It includes the acute and chronic care, the conservative and surgical approach and gives excellent tips which a physician might neglect to mention when advising his patients.

I was very happy to see that he did not attempt to detail a complete exercise program as the lay public too frequently attempts to follow such a program rather than seek proper care and guidance from the physician. So often, exercises are given as a set of instructions which then are improperly performed because they have not been explained or taught by a physical therapist.

Throughout the book the importance of seeking proper medical care for all aspects of the program is emphasized. Also, it makes an outstanding contribution which is frequently forgotten in the care of the back. It discusses the emotional problem in relation to low back pain and treats the problem very realistically. The other aspect, so often forgotten is the relationship of the low back problem to sex. This is dealt with also in a very realistic manner and should be of great assistance to patients with such problems.

I was unable to find any bad points in this book. I feel that it achieved its goal quite well and that the authors knew their subject matter and related it in an enjoyable manner. I would have no objection in recommending this book to my patients with low back pain in order to give them better insight into their problems.

MARGARET KENRICK, M.D.
Director and Chairman,
Department of Physical Medicine
and Rehabilitation
Georgetown University School of Medicine

A Civilian Doctor in Vietnam. FRED GLOECKNER, M.D. Price $5.00. Pp. 123. Philadelphia: The Winchell Company, 1972.

Having served a two month tour of duty as a volunteer physician for Vietnam (VPWN) in 1967, it was truly a pleasure to read Dr. Gloeckner's "A Civilian in Vietnam" written about his impressions in 1966. The narrative about life away from the hospital depicts spicy, probing conversations with military and support personnel in the small town of Ben Tre in central South Vietnam. His role conflict of Physician-Ambassador is torn between the intent of some Americans at Americanizing the Vietnamese and a desire of many natives for Americans to leave the country such that they might resolve their conflict.

The need for physicians during that conflict is extremely acute with only one MD per 100,000 people. As a physician he points out the daily routine on a medical ward in an archaic structure and the problems of treating many tropical and communicable diseases.

Most of the book is not about the medical problems but rather an exploration of military and civilian thoughts about America's role in this war. His background work with the psychodynamics of people make him very sensitive to various reactions about the war and the writing style, though chapterized in diary fashion, is prosaically documented.

It is recommended to all interested in learning some of the reasons why more than 700 physicians from the U.S.A. have volunteered to serve as VP/VNS.

WILLIAM W. FUNDERBURK, M.D.
VP/VN '67

medical annals of the district of columbia

vol. 41 no. 10 october 1972

Review of Medical Microbiology, tenth edition. ERNEST JAWETZ, PH.D., M.D., JOSEPH L. MELNICK, PH.D. AND EDWARD A. ADELBERG, PH.D. Price, 8.00. Pp. 518. Los Altos, California: Lange Medical Publications, 1972.

This Review of Medical Microbiology presents a very fortunate combination of the theoretical and practical aspects of the specialty. It first appeared 18 years ago and is now in its tenth edition, with the same three authors. The appendix on medical parasitology which was introduced into the fifth edition continues, and there is added an excellent chapter on tumor-producing viruses. Earlier editions gave no references to the literature either as a book or as articles and these are now included.

The author microbiologists, as leaders in the field of cytology, present in this volume excellent up-to-date information on cellular metabolism and genetics as derived from studies of microorganisms. This, with very useful chapters on antimicrobial chemotherapy and immunity, make up the first third of the book.

Bacterial species are presented in sixty pages, while viruses are discussed for two hundred pages. Spirochetes, fungi, Rickettsial diseases and parasites are all proportionally presented in a concise and practical manner. Present day nomenclature is followed. The approach is medical with there being included diagnoses and a listing of the clinical features of the diseases, and also statements on treatment. The information on treatment is augmented by the very worthwhile 1971-72 table on drug selections in the chapter on antimicrobial therapy.

The quantitative page differences indicated above for the bacteria and viruses are an asset to the book. The basic information in the first one-third applies mainly to bacteria, so that the chapters on bacterial species have a strong medical approach and are not interrupted by the inclusion of basic information. The virus section follows the same outline, but has little to offer regarding therapy, and is supplemented with basic information to provide a greater understanding of the dissimilarity among viruses. The chapter on oncogenic viruses is very up-to-date and includes a discovery of reverse transcription by Baltimore and Temin.

The chapter on the Principles of Diagnostic Medical Microbiology and the chapter Serologic Diagnosis of Virus Infections are valuable in that they provide an understanding of the specimens to be submitted to the laboratory for diagnosis, and the time and effort required for their processing.

The compact style of the book and the many excellent figures and diagrams, as well as the print, illustrations and binding, with a flexible water repellent cover, are attractive aspects of the book. The fifth edition in 1962 cost $5.50. This tenth edition in 1972 costs $8.00. The present edition has nineteen more pages and four more chapters and many more illustrations. Clinicians, students and technologists will find this book very instructive and useful.

GEORGE P. BLUNDELL, M.D.

The Action of Hormones: Genes to Population. PIERO P. FOA. Price, $34.75. Pp. 574 with illustrations. Springfield, Illinois: Charles C. Thomas, 1971.

Now that the physiologic role and the chemical structure of many hormones have been elucidated, an increasing amount of research and speculation has been centering about their specific mode of action. The action of a hormone can be defined on several levels. Many questions can be asked, and some have been answered. For example, what is the nature of the linkage of a hormone with the receptor cell upon which it reacts? Once contact has been made with the receptor cell, are there certain components within the cell which have a specific affinity for the hormones? Thus, studies with estrogens have revealed specific binding with intra and extra-nuclear macroglobulins, these binding sites being found primarily in the tissues which normally respond to estrogen i.e.: uterus, vagina, pituitary. How do hormones modulate protein synthesis? It is known that insulin acts upon ribosomes, stimulating the incorporation of amino acids to protein.

Is the action of a hormone upon its target cell always direct, or is there an intervening mediator of hormone action? A huge mass of literature in the past half-decade has demonstrated that many peptide hormones activate or inhibit the enzyme adenyl cyclase which is within or adjacent to the cell membrane. This enzyme catalyzes the formation of cyclic AMP (adenosine 3′5′ cyclic monophosphate). Then, cyclic AMP increases in concentration and mediates the action of the peptide hormone, acting as a sort of intracellular messenger. Therefore, some investigators have referred to some peptide hormones as being the first messenger, and cyclic AMP as being the second messenger of hormone action. The determinant of the specificity of hormonal action appears to be due to a variation of the adenyl cyclase molecular configuration from tissue to tissue, permitting chemical interreaction with only that hormone which physiologically should exert an action on the tissue in question.

On a broader scale, hormones exert actions upon groups of cells, tissues, and organs. Thus, insulin produces movements of amino acids from the extracellular compartment into the intact cardiac muscle and into isolated cardiac cells, norepinephrine increases lipolysis of isolated adipose cells etc. On an even larger scale, hormones exert physiologic actions upon the organism as a whole. Thus, growth hormone produces positive nitrogen balance, increases tissue protein and water, and induces a retention of phosphate and potassium.

A recent symposium on the action of hormones was held at the Sinai Hospital of Detroit. Investigators from throughout the country and abroad participated in this meeting, the proceedings of which were edited by Dr. Piero P. Foa (The Action of Hormones, Charles C. Thomas, Springfield, Ill., 1971.) Hormonal action was discussed at a molecular, cellular, organ, organism, and population level. Several of the presentations are extremely well-written. The subtitle of the book is "genes to population," which is intended to call attention to the broad implications of hormonal action, but delivers a bit less than it promises, since only two papers deal with hormonal aspects of population growth. In this rapidly-moving field, some of the findings are already out of date. Nevertheless, although the book offers little of interest to many physicians, it is of considerable value to the investigative endocrinologist, gynecologist, physiologist, and pharmacologist.

KENNETH L. BECKER, M.D.

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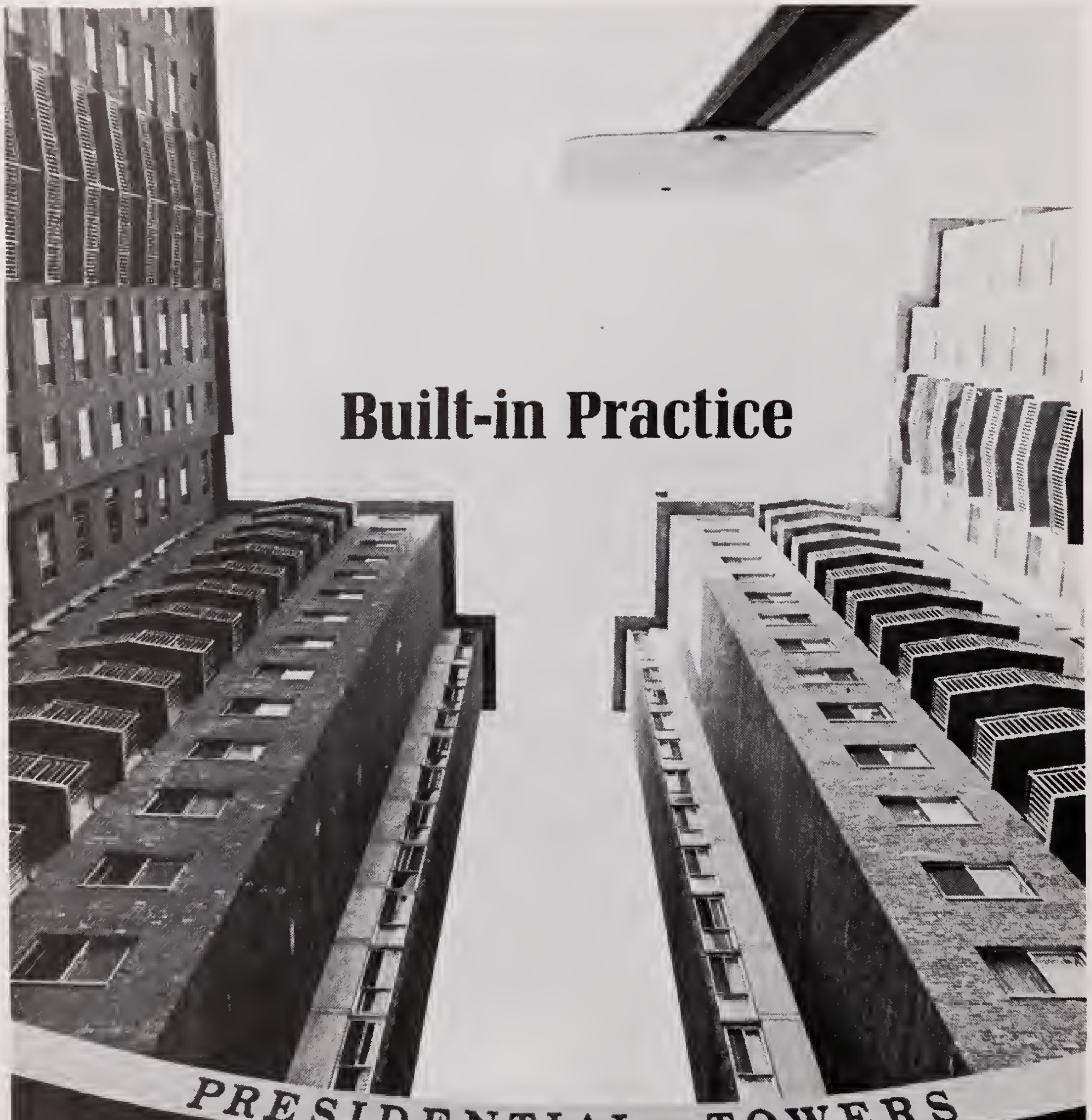

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THE VIEW BOX

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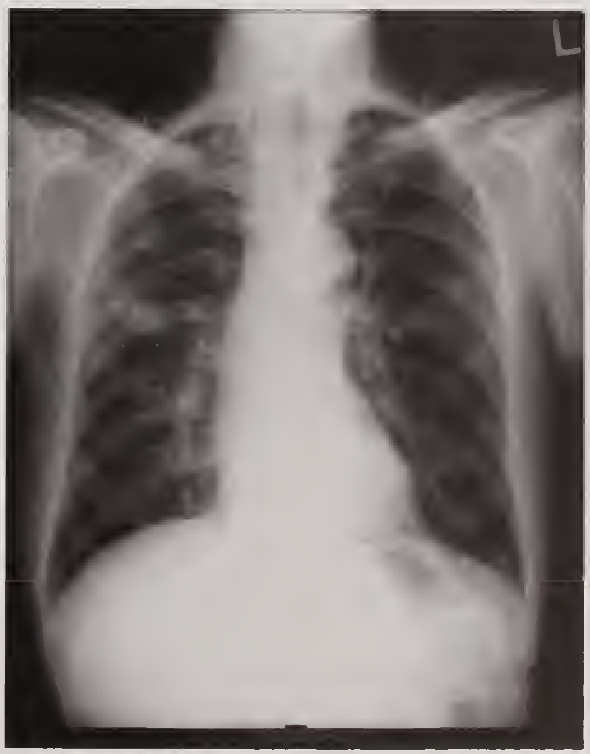

DIAGNOSIS: Pharyngeal (Zenker's) Diverticulum and Aspiration Pneumonia

Note on the PA chest film the air fluid level in the superior mediastinum and the patchy infiltrate in the right lung.

The pharyngeal diverticulum arises between the fibers of the inferior pharyngeal constrictor muscle, usually projects posteriorly, displaces the esophagus anteriorly and can get very large. There is often a history of chronic cough and dysphagia. Barium studies are diagnostic.

Edited by Gunter J Augustin, M.D.
Senior Attending Radiologist, Washington
Hospital Center, Washington, D.C.

obituaries

Robert Emmett Moran, M.D.
1896–1972

Dr. Robert E. Moran, a prominent Washington area plastic surgeon and former physician to the Washington Redskins football team, died July 22, 1972. He resided at 9401 River Road, Potomac, Maryland.

A native of Washington, D.C., he received both his undergraduate and M.D. degrees from Georgetown University. He interned at Columbia Hospital for Women here and at St. Vincent's Hospital in New York City. He was a fellow for four years at the Mayo Clinic in Rochester, Minnesota. He began his practice in General Surgery in the District of Columbia following his residency and became interested in Plastic and Reconstructive Surgery through his treatment of burn victims at Children's Hospital here. Specializing in Plastic and Reconstructive Surgery, Dr. Moran performed an original operation for exophthalmic goiter. He also reintroduced an operation for speech correction for cleft palate.

Physician to the Washington Redskins for more than ten years, he had been on the staffs at Emergency, Georgetown University, Children's, Episcopal Eye, Ear and Throat, Suburban and Sibley Hospitals. He had also been a consultant at Andrews Air Force Base and the Naval Hospital at Bethesda, Maryland.

A Life member of the Medical Society of the District of Columbia, Dr. Moran was a founder of the Eastern Surgical Society, of which he was also a past president, the American Board of Plastic and Reconstructive Surgery, and the International Society of Plastic and Reconstructive Surgeons. He was past president and honorary member of the American Association of Plastic Surgeons, and past president of the Mayo Clinic. He was also a member of the American Medical Association, the American College of Surgeons, and the American Society of Plastic Surgeons.

He is survived by his wife, Jocelyn, of the home; three sons, Dr. Robert E., Jr., of Potomac, Maryland; Peter R., of Vienna, Virginia, and Patrick D. of Potomac; a daughter, Marguerite Dale, of the home; a brother, James O'Donnell Moran; four sisters, Mrs. Mary P. Schuett, Mrs. Catharine M. Burke, Mrs. Margaret M. Hudson and Mrs. Helen M. Wade, and eleven grandchildren.

Oswald E. Roggenkamp, D.Sc.
1908–1972

Dr. Oswald E. Roggenkamp, a specialist in foot surgery, died July 19, 1972. He resided at 212 Church Street, N.E., Vienna, Virginia.

Born in Takoma Park, Maryland, he studied at the Washington Missionary College and the Illinois College of Chiropody and Foot Surgery, from which he graduated in 1934. He took his postgraduate training at the Ohio College of Podiatry.

An Associate member of the Medical Society of the District of Columbia, Dr. Roggenkamp was also a member of the National Association of Chiropodists, the American Podiatry Society and the American College of Podiatric Medicine. He was a fellow of the American College of Foot Surgeons and the American Academy of Podiatrists.

He is survived by his wife, Dora Caylor Roggenkamp, who had most recently been Assistant Executive Director of the Medical Bureau of the Medical Society of the District of Columbia, and had been employed by the Medical Society of D.C. for more than 20 years, of the home; a son, Clyde Loren Roggenkamp, of Springfield, Vermont; a daughter, Marilyn Alice R. Cole, of Wheaton, Maryland; his mother, Alice W. Roggenkamp, and a stepson, T. C. Caylor of Sterling Park, Virginia; a brother, Wilbur Roggenkamp, and four grandchildren.

Elizabeth Kittredge Whittemore, M.D.
1889–1972

Dr. Elizabeth Kittredge Whittemore, a Washington area physician who was active for many years in the Child Welfare Clinic and Planned Parenthood Program here, died July 23, 1972. Known professionally as Dr. Elizabeth Kittredge, she was the widow of Herbert Lucius Whittemore. She resided at 3906 McKinley Street, N.W.

Born in Westford, Massachusetts, she was a graduate of Vassar College and received her M.D. degree from Johns Hopkins University. She also studied at Harvard University and held a masters degree from Columbia University. She was the first woman intern in general medicine to serve at Saint Elizabeths Hospital here, and she served her residency at Vassar College.

Dr. Kittredge had served with the Board of Public Welfare and the Child Welfare Clinic at Children's Hospital, and had also worked at the Receiving Home for Children. She had also supervised a number of Planned Parenthood clinics as well as prenatal clinics for the Health Department.

A Life member of the Medical Society of the District of Columbia, she also held membership in the American Medical Women's Association and the District of Columbia Women's Medical Association, of which she was a past president. She was Editor of the American Medical Women's Association Journal for ten years. She was also a member of the American Medical Association.

Dr. Kittredge is survived by a son, William Kittredge Whittemore, of Cleveland; a daughter, Mrs. James E. Simon, of Washington, D.C., and five grandchildren.

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president's message

The People's Republic of China
A Dialogue With Visiting Physicians

arrell C. Crain, M.D.

The recent visit of physicians from the People's Republic of China to our country demonstrated anew the respect with which American medicine and American physicians are held in this country and abroad. It also emphasized the role and the responsibility of our profession in promoting friendship among the people of the world. Everyone who met the visitors was impressed by their pleasing personalities as well as their openness and desire for frank discussions. One thing seemed to stand out: the similarity of physicians regardless of their country of birth and training, and their mutual concern for solutions to problems of the sick.

Physicians old enough to have served overseas in World War II may recall the difference between medical and military men in their approach to captured documents: doctors were searching for information regarding new and different approaches to the treatment of disease; generals were looking for enemy battle plans. So, as the cold war in Asia thaws, it is the physician who is looked to for help in healing the wounds of distrust and suspicion.

It seems unfortunate that the day has passed when all medical writings were in a common language. Probably the time formerly devoted to learning Latin is better spent on other subjects in the medical curriculum, but the language barrier is still present. In this regard, the Chinese physicians exhibited an amazing knowledge of what American physicians are doing. Despite the ban on interchange of literature, there can be no doubt that the contents of many American medical journals are well known to Chinese physicians. With the resumption of communications it should not be too long before we begin to have insight into their investigations also. Since dialogue with our Soviet colleagues has already been established, future cooperative ventures around the world could well usher in an era of unprecedented medical progress.

Current Practice of Biliary Surgery (Kune) Reviewed by Richard L. Flax, M.D.

Review of Medical Pharmacology (Myers, Jawetz and Goldfien) Reviewed by Maurice Van Kinsbergen, M.D.

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Yes, despite all the attacks our profession has endured, it retains a unique position in our civilization, as it has in others before us. Let us ever remain above the struggles which seem about to destroy many other institutions in our society. So shall we maintain the heritage of Aesculapius and Hippocrates and help bring about that better world everyone is seeking.

Darrell C. Crain

"But where are the nine?"

Luke 17:17

This well-known verse from the New Testament brings to mind the healing of the ten lepers by Christ and the subsequent return from the temple of only one of them to give thanks. It stands as a classic tale of ingratitude and selfishness.

But what does this have to do with you as a member of this Medical Society? It can, in fact, be applied, mutatis mutandis, to the situation which exists in the medical community in this metropolitan area.

By that I mean the general lack of appreciation for the amount of time, effort and thought that is expended by a small number of members in accomplishing the work of the Society. The entire membership benefits from their labors, and yet how many indicate their appreciation?

In point of fact, when the President-elect sends out his call for committee members he will get, on an average, some 200 replies from a membership of 1,700 plus. "But where are the nine?"

When one sits at meetings of hospital staff committees, Blue Shield committees and Medical Society committees, one is not overwhelmed with a sea of unfamiliar faces. No, indeed. The same faithful, committed, hard-working ten per cent are seen at those meetings. "But where are the nine?"

Don't write off their participation as an ego-trip for them. Many of them dislike the work but do it because they are convinced of its importance. Don't assume that they are medical politicians with aspirations to local or national office. Most of them would rather be sitting in their offices practicing medicine just as you or I.

Don't assume that they get a sense of power from committee work. Each one of them could truly be an autocrat in his own office and avoid the frustrations of majority decisions in committee work.

No, all those self-serving arguments only attempt to obscure the fact that the many have been willing to let the few do the work.

If you truly care about the future of medical practice (whichever form you favor), if you have been unhappy about decisions and postures assumed by the Society, and if you really want the voice of the Medical Society to represent the views of the rank and file, then participate.

Participate in your section meetings, participate in Society committee meetings, participate in the form of letters to the MEDICAL ANNALS, or to the Executive Director, the President or the Chairman of the Executive Board.

Then, when they ask "But where are the nine?" they will not be talking about an unconcerned you.

R.W.L.

Review of Thrombosis of Abdominal Aorta with Report of Two Asymptomatic Cases

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Thrombosis of the abdominal aorta causes two clinical syndromes. One consists of excrutiatingly severe pain in the feet and legs of abrupt onset associated with circulatory disturbances in the lower extremities that rapidly progress to ischemic necrosis. The other, which is more common and occurs more often in men, consists of progressive fatigability, intermittent claudication, and sexual impotence that is gradual in onset and may last for years. The latter is known as the Leriche syndrome. Rarely, in contrast to these two syndromes, thrombosis of the abdominal aorta may be altogether asymptomatic. Two cases of this kind, both occurring in women, are therefore of interest.

Report of Cases

Case 1. The patient was a white woman 61 years old who was awakened by pain in the neck and shortness of breath that were followed by diaphoresis, nausea, and vomiting. She was admitted to the hospital with a diagnosis of acute myocardial infarction. She had been well until five years previously when angina pectoris developed. Examination at that time revealed a palpable pulse only at the right wrist. The angina recurred and was treated with nitroglycerine. She had not had orthopnea, dependent edema, or intermittent claudication.

Physical examination revealed a pulse only at the right wrist. The pulse rate was 100 and the temperature was 37° C. The blood pressure was estimated by palpation to be 100 mm Hg. Rales were audible over both lower lung fields. A systolic ejection murmur was heard along the left sternal border. The abdomen was not remarkable. The deep tendon reflexes were physiologic.

The urine disclosed many leukocytes. The blood was normal except for a leukocytosis of 12,900 with a moderate neutrophilia. The serum glutamic transaminase was 225, the lactic dehydrogenase was 1275, and the creatine phosphokinase 117 upper 100 ml. An X-ray film of the chest disclosed pulmonary edema. An electrocardiogram indicated subendocardial injury. An ultrasonic detector revealed no pulse in the dorsalis pedis and posterior tibial arteries and only a slight pulse in the subclavian, radial, iliac, and femoral arteries. The patient was given oxygen, digitalis, and diuretics. Congestive failure increased and death occurred after 14 days in the hospital.

Autopsy. The heart weighed 285 gm. The left ventricle and interventricular septum revealed recent infarcts. No mural thrombi were found. There were also old infarcts of the wall of the left ventricle, calcification of the mitral annulus, and marked atherosclerosis of the coronary arteries. The abdominal aorta and segments of the iliac, femoral, subclavian, innominate, and common carotid arteries were somewhat smaller than normal, and the lumens were occluded by recent, organizing, and healed thrombi. Various degrees of intimal fibrosis were also present. The abdominal aorta is shown in figure 1. Additional findings included edema of the right and left lungs, bilateral hydrothorax, and an old infarct of the right occipital lobe of the cerebrum.

Histologic examination of the aorta and large arteries revealed pronounced deposits of fatty material with cholesterol clefts, marked subintimal fibrosis, and canalized thrombi filling the lumens. Inflammation was inconspicuous, giant cells were seen only sparsely in the regions of cholesterol clefts, and the elastica was for the most part intact. Calcification was present focally at the junction of the subintimal connective tissue and media. The adventitia was not remarkable. The aorta with intact media and organized thrombus is shown in figure 2.

Case 2. The patient was a white woman 73 years old who experienced nausea and vomiting after an evening meal. This was soon followed by acute shortness of breath which necessitated admission to the hospital. There was no chest pain or diaphoresis. She had been taking a cardiac medication, presumably digitalis, for the past 2 years. There was no history of intermittent claudication, orthopnea, or nocturnal dyspnea.

The temperature was 37° C., the pulse rate 140, the respiratory rate 34, and the blood pressure 150/100 mm Hg. Rales were audible over the lower two thirds of both lung fields. The heart was slightly enlarged to the left. No murmurs were heard. The abdomen was not remarkable. Pulses were palpable in the upper extremities, but the femoral, popliteal, and dorsalis pedis pulses were not palpable. The deep tendon reflexes were absent in the right leg and were physiologic elsewhere.

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The hemoglobin was 11.8 gm., the hematocrit 37.8%, and the leukocytes numbered 38,800 with 55 segmented granulocytes, 22 band cells, 4 juvenile cells, 15 lymphocytes, and 4 monocytes. An EKG revealed atrial fibrillation that changed to complete heart block. Respiratory difficulty worsened, and the patient died on the day after admission.

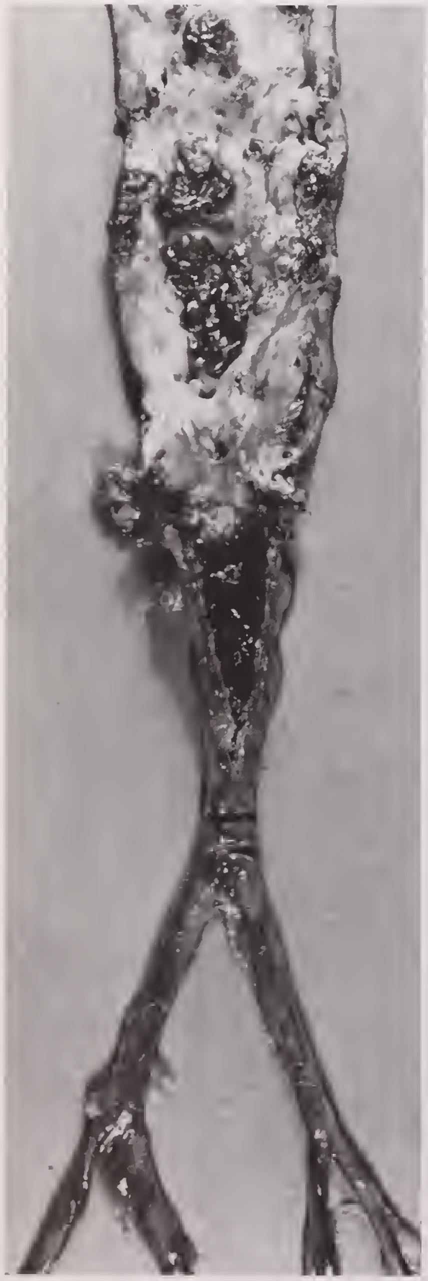

FIG. 1. Case 1. Thrombus of abdominal aorta extending into common iliac arteries. Atrophy is conspicuous in occluded vessels.

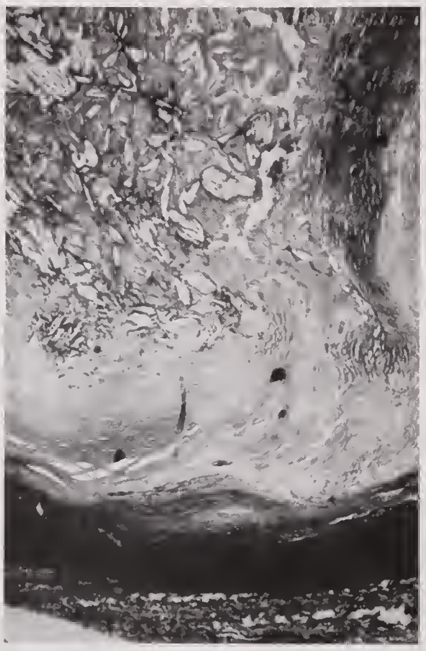

FIG. 2. Case 1. Histologic section of aorta showing intact media and organized thrombus with cholesterol clefts.

Autopsy. The heart weighed 550 gm. The left ventricle disclosed an old infarct, and the mitral valve revealed healed rheumatic endocarditis with slight stenosis. No mural thrombus was found. Arteriosclerosis in the coronary arteries was slight. The abdominal aorta was occluded by a recent and organizing thrombus that extended proximally to the superior mesenteric and right renal arteries and distally to the right and left common iliac arteries. The cut surface of the abdominal aorta disclosed a solid thrombus that had undergone partial decolorization (fig. 3). Additional findings included chronic pyelonephritis of the kidneys, bronchopneumonia of the right lung, and an old infarct of the spleen.

Histologic examination of the aorta revealed a large recent thrombus composed principally of fibrin. At the margins granulation tissue was evident with fibroblasts extending into the fibrin mass and covering the intimal surface. The media was intact, and the intima revealed only moderate atherosclerosis; calcium was not present, cholesterol clefts were scarce, and only a small number of lymphocytes were present in the foci of sclerotic change. The aortic wall with recent thrombus and organizing margin is shown in figure 4.

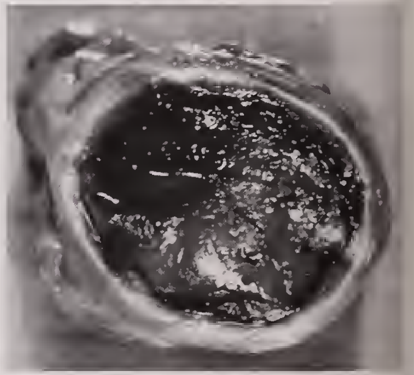

FIG. 3. Case 2. Cross section of abdominal aorta with lumen occluded by large recent thrombus.

Comment

Although it appears astonishing that so large a vessel as the abdominal aorta

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may be entirely occluded without causing symptoms, several cases have been described.[1-6] In most of these the patients were afflicted with slowly progressive disease and were bedridden for long periods. Neither condition applies to the present cases.

Historical Data

Aortic thrombosis was mentioned in the English literature in 1814[7]. In those times it was recognized by agonizing pain in the legs and was regularly attributed to disease of the heart. Moreover, treatment was unavailable and fatality was the certain outcome. Since then a wider understanding of the condition has evolved, and the pattern of this evolution provides a microcosm of medical progress. It begins with the bleak days of blisters and bleedings and progresses through several levels of advancing knowledge to the present time of expanded clinical awareness, sophisticated diagnostic techniques, and curative surgical procedures. Review reveals the rate of that progress and the significant steps along the way.

FIG. 4. Case 2. Histologic section showing recent thrombus, organizing margin, and intact wall.

In 1814 Graham[7] described a case of "obstructed aorta" due to a thoracic coarctation in which the patient was treated with blisters and bleedings. Therapeutic futility was indicated by his last sentence, "The practical inferences from this are obvious, for it is clear, that under this and all other incurable organic lesions of vital parts, life can be protracted and suffering alleviated, only by avoiding exposure, fatigue, emotions of the mind, and stimulating diet." The patient, a 14-year-old boy, died. In discussion of this case, Graham mentions a museum specimen ". . . where the aorta is plugged up by a laminated coagulum just above the bifurcation of the iliacs, into both of which this substance extends." The owners of the museum were unable to find any account of this case in the papers of a Mr. Allan Burns, to whom the specimen belonged. This appears to be the first notice of aortic thrombosis in English. According to Burt, Learmonth and Richards,[8] however, foreign reports of the condition had been registered by Severinus in 1632, and by Fantonus in 1679. Early American accounts were recorded by Wilbur in 1857,[9] and by Osler in 1887.[10] In 1899 the condition was discussed as an entity by Welch,[11] who collected 59 cases, many from the foreign literature. He pointed out that most were associated with heart disease, had an acute onset, and were presumed to be embolic. Priapism was noted to be an "exceptional symptom" of the condition. There followed single case reports by Fawcett,[12] Hawkins,[13] Daggett,[14] and Wylde.[15] In 1928, Banowitch and Ira[16] reported five of their own cases, and Moschowitz[17] and Rothstein[18] stated that the condition also occurs in infants. Variability of symptoms was emphasized by Lueth,[19] Gross and Philips[3] cited an asymptomatic case, and Ravdin and Wood[19] performed one of the earliest successful embolectomies. In 1940 Leriche[20] described a new syndrome in which the onset was gradual, the symptoms mild, and the survival long. It was more common in men and was characterized by fatigability of the lower extremities, intermittent claudication of the thighs, and atrophy of the calves. In addition, there was sexual impotence with failure to maintain an erection. This syndrome was attributed to atherosclerosis of the abdominal aorta with gradual thrombosis, whereas the sudden-onset syndrome was presumed to be embolic. Later it became evident that the mode of onset does not reliably indicate the nature of the pathologic process,[21-23] and, as experience increased, the frequency of embolism diminished. With experience and experimentation, methods of treatment were devised, and these included sympathectomy, aortic ligation, embolectomy, thrombo-endarterectomy, aortectomy, and by-pass grafting. The most sophisticated was resection of the abdominal aorta and replacement of that portion with a prosthetic graft. Under optimal conditions treatment now provides for immediate restoration of the circulation to be lower extremities.

Demography

In a review of reports citing a total of 33,226 autopsy examinations, Starer and Sutton[6] found 50 cases of aortic thrombosis and indicated an incidence of about 0.15 [illegible] An approximately similar experience was noted by Barnett, Moorman and Merrick[24] who found 10 cases in 4,394 necropsies Gross and Phillips[3] observed seven cases in 5,350 autopsies and suggested an incidence of slightly more than one per 1,000 examinations Siegel and Garvin[25] found 11 cases in

6,547 autopsy examinations, and Lueth[1] cited one case in 1,047 "post mortems." Beckwith and colleagues,[26] however, regarded the condition as common, diagnosing 65 cases in a five-year period.

Most of the patients are males, as evidenced by the preponderance of them in various series; 90%,[24] 89%,[27] 84%,[28] 75%,[6] 74%,[29] and 63%.[30] In one report, however, there were 10 females and only four males,[31] and in another account of five patients all were females.[16] It was stated by Greenfield,[5] and by Leriche,[4] that embolism occurs equally in the sexes, whereas thrombosis is twice as frequent in males.

In has been emphasized that aortic thrombosis is a disease of the middle years rather than of old age.[6] The average age is usually in the early fifties,[21,22,25,27] and the range of age in one series[28] extended from 27 to 57 years, and in another from 37 to 78 years.[6] All of the 14 patients in the report of Crane[34] were in their forties and fifties. The oldest patient found in this review was 81.[32] The condition does occur in infancy, however, and the ages include nine days,[30] 10 days,[17] 10 days,[18] and 11 months.[34] A familial occurrence was noted by Harrison, Keshishian and Gerwig;[35] the patients were all males, two brothers and a cousin, and four others in the family had clinical evidence of the same lesion.

Clinical Aspects

In the early reports, leg pain of abrupt onset was the main clinical feature.[11,16] By 1906 Fawcett[12] regarded the clinical events as forming a "well-marked group"; they included "pain in the legs, loins and the abdomen, a sudden paraplegia of the flaccid variety, and, as the most important diagnostic symptom of all, absence of pulsations in the femoral arteries, with cold livid extremities in which gangrene may develop, should the patient live." Lueth[1] described the pain as "severe to agonizing," arising in the loins, and extending down both legs. Absence of pulsation in the lower abdominal aorta was noted by Starer and Sutton[6] and by Bell.[22] The clinical syndrome was thus discrete and sharply defined, although occasional variations were noted. Paresthesia about the rectum in the form of formication extending down both legs was described by Ravdin and Wood,[19] numbness in the legs was noted by Hawkins,[13] and renal failure from extension of the thrombus to the renal arteries was observed by Potter and Hopkins.[36] The generally accepted clinical picture, as summarized by Gross and Philips[3] in 1940 included severe pain in both legs of sudden onset accompanied by sensory loss, a-reflexia, numbness, coldness, pallor, cyanosis, and loss of pulsations followed by paraplegia, shock, gangrene and death. The five diagnostic "P's" of Albright and Leonard[37] included pain, pallor, paralysis, pulselessness, and paresthesia.

After this early characterization, other aspects were described. They were, especially, a slow and gradual onset with claudication as the main manifestation.[16] Lueth[1] also noted a slow onset, and when ischemic necrosis developed, he related its extent to the adequacy of the collateral circulation. Gross and Philips[3] stated that the main collaterals carrying blood around the thrombus were the internal mammary to the inferior epigastric, the inferior mesenteric to the internal pubic, and the lumbar branches of the aorta above the block to the branches of the internal iliac arteries below. It remained for Leriche,[20] however, to emphasize limp erection along with extreme fatigability of the lower extremities, extensive atrophy of the lower limbs, and intermittent claudication with absent femoral pulsations, as the main clinical features of the chronic process. He further pointed out that the gradually developing condition is more frequent than the acute syndrome, occurs more often in young men, and may be well borne for five to 10 years. Greenfield[5] gave a comprehensive clinical account, although he did not mention impotence as a symptom. Elkin and Cooper[38] noted that the average duration of complaints in 10 patients with "insidious thrombosis" was seven years, and Beaconsfield and Kulin[28] described 35 cases of their own, in one of which the duration was 21 years. DeWolfe and colleagues[27] emphasized that the claudication may appear in the hips, buttocks, or lower back, and should not be mistaken for orthopedic disturbance of the hips or lumbosacral spine. Beckwith, Huffman, Eiseman, and Blount[26] reported that 23 of their 65 patients admitted to decreased potency, whereas Starer and Sutton[6] found that only four of 24 males would acknowledge it. Beckwith and colleagues[26] described trophic changes of the skin including atrophy, disappearance of hair, and decreased growth of toe nails. They further pointed out that while the femoral pulses were always absent, collateral supply occasionally provided a weak posterior tibial or dorsalis pedis pulsation. Starer and Sutton[6] found no menstrual abnormalities in four women in whom aortic thrombosis developed prior to the menopause.

Distinguishing sign from symptom, Ortner and Griswold[39] more recently characterized the clinical aspects of the slow-onset syndrome. The latter listed as easy fatigability of the lower extremities, intermittent claudication of the legs, pain in the calves, thighs, hips or lower back, coldness of the feet, and inability to maintain an erection. The signs listed as absent pulsations in the femoral arteries and terminal aorta, pallor of the legs and feet even when dependent, atrophy of the musculature of the lower extremities, and trophic changes of the skin that gradually progress.

Although emphasis thus tended to shift from the acute-onset syndrome to the cases with gradual onset, other patients were reported that were asymptomatic.[1-6] These were for the most part associated with advanced disease and prolonged restriction to bed.[6] In one case the diagnosis was made by physical examination, although the patient, with Marfan's syndrome, was admitted to the hospital because of lens dislocations.[40] In the case of Price and Wagner,[41] the patient had no pain and complained of numbness, tingling, and weakness of the lower extremities only after "prolonged effort".

Morphology and Etiology

The thrombus usually lies distal to the ostium of the inferior mesenteric artery and extends to the iliac bifurcation. It undergoes organization and subsequent fibrosis. As this occurs, atrophy develops, and in one case the obstructed vascular segment was reduced to a narrow fibrous cord in a period of eight months.[42] The first portions of the iliac arteries are also usually involved, but it is not clear whether the process begins in those vessels and extends to the aorta, or the reverse.[4] When the thrombosis is gradual, the iliac arteries also undergo atrophy and become slender "stick-like" structures.[4] This change is seen in figure 1. The occluded segment of aorta is markedly atherosclerotic. The process tends to be sharply delimited, however, and the vessel above and below the thrombus is often much less affected.[43] The distal consequences of the aortic obstruction include ischemic necrosis that is widespread in the lower extremities when the onset is abrupt, and ascending in pattern after long periods of trophic change when the onset is gradual. Regardless of the nature of the onset, however, all patients eventually develop ischemic necrosis unless treatment is given.[5]

The thrombus may extend cephalad to block the inferior mesenteric artery. Although this is infrequent and may account for abdominal pain,[22] it is usually asymptomatic because of collateral flow through the superior mesenteric to the left colic and superior hemorrhoidal arteries.[6] Proximal extension to a renal ostium with infarction of the kidney is also uncommon and occurred in only two of 65 cases in one series.[26] Several isolated examples have been described,[10,12,44,15,32,5,18] however, and it occurred in two of the 11 cases noted by Siegel and Garvin.[25] Renal failure, reversible by prompt therapy, has also been reported.[36] In some patients the thrombus occluded the superior mesenteric artery and caused necrosis of the bowel.[13,44] In others, it extended to the celiac axis.[15,44] Aneurysm of the aorta, diabetes mellitus, and neoplastic disease are infrequent and do not appear to be predisposing conditions. Hypertension occurred in 32 of the 65 cases described by Beckwith and colleagues.[26] With the slow-onset syndrome, death is seldom attributable directly to the thrombus and is usually due to atherosclerotic disease of the coronary, renal, or cerebral vessels.[26]

Concern for cause was expressed by Wilbur's report of 1857 wherein he asks, "How did the disease of the heart, or other disease, produce these plugs?'"[9] In those times an abrupt onset was characteristic, and embolism was presumed more often than thrombosis. Welch,[11] for example, noted in his review of 59 cases that mitral stenosis was present in 34%, and he speculated "without much confidence" that 45 of the 59 cases were embolic in nature. Banowitch and Ira[16] also observed that over half of the cases they reviewed were associated with heart disease, especially mitral stenosis with auricular fibrillation, myocardial infarction with mural thrombosis, and bacterial endocarditis with valvular vegetations. By 1940, however, the syndrome of slow onset was more widely recognized, embolism less often presumed, and Lueth[1] and Leriche[4] both emphasized that pronounced atherosclerosis of the abdominal aorta is the principal cause. Atherosclerosis was regarded as the cause of thrombosis in 28 of the 35 cases reported by Beaconsfield and colleagues,[28] and in all seven of the cases described by DeWolfe et al.[27] Beckwith, Huffman, Eiseman, and Blount[26] made a similar emphasis, and Starer and Sutton[6] pointed out that only seven of their 32 patients had heart disease.

In addition to cardiac disease and advanced atherosclerosis, aortic thrombosis has been attributed to or associated with typhoid fever,[10] bronchogenic carcinoma,[1] metastatic carcinoma of the spine,[3] tuberculosis,[5,45] massive necrosis of the liver,[5] volvulus of the intestine,[5] vertebral exostosis,[32] Addison's disease,[45] Buerger's disease,[46] Marfan's syndrome,[40] saccular aortic aneurysum,[22,23] poliomyelitis,[6] syphilitic aortitis,[28,5,44] trauma,[28,47,48,42,49] a bullet embolus,[50] and intravenous pyelography with compression of the abdomen.[51] It has coexisted with horseshoe kidney.[52,53] In infants, diarrhea,[33] pneumonia,[34] and suppurative inflammation of the umbilicus,[17,18] have been associated conditions. The infant described by Rothstein was afflicted with Down's syndrome.[17]

Diagnosis

Diagnosis has not been difficult because of the characteristic agonizing pain in the legs with abrupt onset, or claudication of the thighs with gradual onset, both accompanied by pulseless lower extremities. To this has been added absence of abdominal pulsations from the umbilicus down[54] and palpation of the margin of the thrombus in the midabdomen.[6] DeWolfe et al[27] claimed that claudication of the hips is pathognomonic of arterial thrombosis proximal to the inguinal ligament. A plain film of the abdomen frequently reveals calcification of the aortic wall.[6] With the advent of aortography, diagnostic precision was achieved.[41,4] Shapiro[55] emphasized that the method would identify the site of the thrombus, the extent of the blockage, and the state of the collateral circulation. Moreover, the procedure is useful in distinguishing aortic saddle embolus from atherosclerotic thrombosis.[23] Aortography also discriminates between thrombosis of the abdominal aorta and bilateral thrombosis of the iliac arteries, although, as Beaconsfield et al[28] pointed out, the distinction is somewhat academic since the two conditions are treated in the same way. Danto and colleagues[23] recommended that the injection be made well cephalad of the renal ostia in order to avoid embolization or dye-nephrotoxicity. In one case the procedure precipitated a total aortic occlusion that was attributed to vasospasm.[35] Differential diagnosis includes Buerger's disease of the leg arteries and phlebothrombosis of the leg veins. In the former, pulses are often palpable, the onset is frequently unilateral, and impotence is not a symptom.[46] With phlebothrombosis the extremities may be cyanotic and swollen, but the skin is warm, the pulsations are intact, sensory function is unchanged, and the veins are often tender to palpation.[18]

Treatment

In the beginning, aortic thrombosis was regarded as a medical curiosity and little attention was given to therapy.[9-13] The reason was that heart disease was almost always associated, the condition was almost always sudden in onset and rapidly progressive, and the outlook was essentially hopeless. This bleak prospect was evidenced by the report of Banowitch and Ira[16] who collected 105 cases from the literature and found that all had died within a period of "a few" to 44 days. Nevertheless, they suggested massage of the lower abdomen to dislodge the clot to one or both legs, admitting that the procedure "may require amputation later." Embolectomy was subsequently attempted, and Neuhof[21] noted in 1932 that the method is formidable, the patient is usually in severe shock, and the circulation is sometimes restored by simply "awaiting developments." He accordingly declared that it is questionable "whether operation should ever be attempted on the abdominal aorta for an embolus lodged at the site." This grim view was not entirely without evidence; by 1935, Rothstein[18] was able to cite 123 case reports of which 112 or 91% were fatal. Of the 11 that lived, three improved spontaneously, three required amputation of the leg, and five survived the procedure of embolectomy. The mortality of the aortic operation, however, was 68%. Early details of the operation, and suggestions for improvement were recorded by Kerr.[56]

One of the first American operative successes was that of Ravdin and Wood in 1941.[19] They opened the aorta, inserted a Babcock vein-stripper, and fragmented the thrombus. The blood flow was immediately restored and the patient was able to leave the hospital within two months. The authors also recommended sympathectomy to relieve the vascular spasm, heparin to prevent propagation of the clot, and pressure-suction therapy to promote circulation in the legs.[19] In 1943 Murray[57] reported five cases of successful aortic embolectomy, and by 1947 embolectomy was established as the preferred method of treatment.[58] Nevertheless, in 1952 Pool and Farrar[59] were able to find only 32 cases in which the procedure had been successful. In the following year Lord and Burke[60] described three additional cases, gave the details of the procedure, and suggested the advisability of "auricular appendectomy" and mitral commissurotomy after evaluation of the patient by cardiac catheterization. Embolectomy had been successful in cases of traumatic thrombosis of the aorta[47-49] and in patients with associated paraplegia and renal failure.[36] More recently Crane[31] stated that the circulation can usually be restored if the operation is performed within six to 10 hours after the thrombotic event. He observed that aortic embolism is often heralded by small embolic episodes, and suggested prophylactic replacement of the mitral valve for the patient with mitral stenosis who has had such a forewarning event. In order to diminish the chance of recurrent thrombosis, Beckwith and colleagues,[26] and later Bell,[22] recommmended leaving the thrombus in place and by-passing the occluded segment with a prosthetic graft.

The earliest methods for the treatment of the slow-onset syndrome were devised abroad, and especially in France.[4] The first American success, however, appears to be that of Holden[54] who was unaware of these reports. In 1946 he performed a bilateral lumbar sympathectomy on two middle-aged men with pulseless extremities who complained of claudication, impotence, and atrophy of the leg muscles. Both improved. Two years later Leriche[4] gave an English account of his extensive experience. He had achieved success in several patients with "ganglionectomy" and stated that the method should be chosen according to the condition of the patient. For the optimal patient he recommended resection of the thrombosed segment followed by sympathectomy; for the "medium" patient he suggested sympathectomy with optional aortectomy, and for the "poor" patient he advised sympathectomy alone.[4] Elkin and Copper[38] also selected the method according to the individual, and chose among sympathectomy, aortotomy, terminal aortectomy, and amputation of the extremity in the event of ischemic necrosis. Boyd[46] recommended adjunctive ligation of the inferior vena cava, but this was not subsequently adopted. The improvement of prognosis achieved by these procedures was emphasized by Barnett et al[24] and by Leriche and colleagues.[61] In 1952 Wylie[62] utilized thrombo-endarterectomy, achieved patency of the blocked vessel in 25 of 26 patients, and restored the peripheral circulation in 19. West and colleagues[63] also reported success with this technique, as did DeBakey et al[43] when the procedure was restricted to patients in whom the aortic lumen was widely patent above and below the thrombus.

It was foreseeable, however, that resection of the blocked segment and replacement of the defect with a graft would ultimately be the method of choice. Leriche[4] is reported to have forecast this as early as 1923, and Oudot[28] is credited with the achievement of this goal in 1951. Two years later Oudot and Beaconsfield[64] successfully repeated the procedure in five patients, and trophic changes of the skin and persistent ulcers of the legs promptly healed. Beaconsfield and Kunlin[28] further applied the method and included sympathectomy to improve the circulation of the legs. As resection and graft replacement became possible, satisfaction with thrombo-endarterectomy diminished, and several spoke against this method.[27,35,26] When the atherosclerotic process is both widespread and pronounced, Louw[65] suggested an axillary-femoral by-pass graft. The surgical experience for the gradual-onset syndrome was recently reviewed by Spiro and Cotton[29] who declared the preferred procedures to be thrombo-endarterectomy or resection with plastic prosthesis replacement. It was their judgment that aortography would reveal the extent of the thrombus and that resection would be well tolerated if the profundus branch of the femoral artery were patent and well visualized. In validation of this, they cited a 3% operative mortality for their last 32 patients.

Prognosis

In the treated patient, death is seldom due to the aortic lesion. Beckwith et al[26] found hypertension or significant cardiac, renal, or cerebrovascular disease in 56 of 65 patients, and asserted that "aorto-iliac thrombosis is but one manifestation of a generalized process, atherosclerosis." Starer and Sutton[6] emphasized that useful life for years is common, and DeBakey and colleagues[45] restored pulsatile circulation in 95% of their patients. Pilcher, Barker, and Cannon[30] reported that in 69 patients with aorto-iliac thrombosis treated by endarterectomy the vessel was patent in 89% five years later and in 69% 10 years after the operation.

Concluding Comment

In the past 160 years the understanding of aortic occlusion has expanded from a single, catastrophic, agonizing, and hopeless condition to two clinical syndromes, each recognizable by unmistakable clinical signs and each amenable to curative surgical procedures that provide for comfort and long life. One is uncommon—embolic, abrupt in onset, and due to disease of the heart, while the other is comparatively common—thrombotic in nature, gradual in onset, and due to atherosclerosis of the aorta. Since the earliest recognition of this condition the lesion has remained constant. This constant morphology is reflected in Charcot's comment, "Disease is from old and nothing about it has changed. It is we who change as we learn to recognize what was formerly imperceptible."[66] This thought may be extended to the treatment of what was formerly beyond therapy. Thus, while pause may be taken on the plateau of surgical success, the distant objective is, as it always has been, the treatment or the prevention of the atherosclerotic process itself.

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Physiological Rationale for Medical Treatment of Duodenal Ulcer

WILLIAM O. DOBBINS, III, M.D.*

The single best discussion of the management of duodenal ulcer was published by Grossman in 1961.[1] Since then little new information has been added to our knowledge of the treatment or of the pathogenesis of duodenal ulcer. Duodenal ulcer is more likely to occur in males, but the incidence in females is rapidly increasing as women's liberation takes full effect. There is a familial tendency to the development of duodenal ulcer which is probably related to the inheritance of parietal cell-mass.[2] There seems also to be some relationship of constant stress to the development of active duodenal ulcers. A recent report by McGuigan and colleagues[3] shows that gastrin may play a significant role in the hypersecretory state which frequently characterizes duodenal ulcer disease. The average duodenal ulcer patient secretes approximately twice as much hydrochloric acid as the average "normal" patient, and massive hypersecretion of acid seen in patients with hypergastrinemia of the Zollinger-Ellison syndrome is associated with a "malignant" duodenal ulcer diathesis.

There are virtually no control studies concerning the treatment of duodenal ulcer disease. Some control studies have suggested that long-term administration of anticholinergics and a new drug, Depepsin (not yet approved by the FDA for use in this country), are very effective in the treatment of duodenal ulcer disease.[4] Other studies, however, fail to confirm the findings or dispute their rationale.[5,6]

Nevertheless, duodenal ulcer is an epidemic disease, and even though we lack objective, controlled, therapeutic trials, we must make recommendations for treatment based upon the little physiological data available to us. There are three statements which can be made categorically concerning duodenal ulcer disease: 1. Duodenal ulcer does not occur in the absence of acid secretion by the stomach ("no acid-no ulcer"); 2. Intensive antacid therapy invariably relieves the pain of duodenal ulcer; and 3. These ulcers recur with a distressing frequency ("once an ulcer-always an ulcer"). Thus, the keystone of medical treatment of duodenal ulcer is intensive antacid therapy. Because there are no controlled clinical trials that convincingly show that antacid therapy speeds the rate of healing of duodenal ulcer, one cannot categorically recommend one antacid over another. It is nevertheless important to give an antacid that reacts rapidly with hydrochloric acid, and calcium carbonate is by far the single most effective commonly used antacid.[1] Calcium carbonate is the standard ingredient of Tums and Dicarbocil and can also be purchased as a powder in an even less expensive form. A major limiting factor in the use of calcium carbonate is that approximately 5% of patients develop hypercalcemia when treated with this drug. Thus, a number of aluminum- and magnesium-containing compounds are available which seem to be quite safe, although far more expensive. Calcium carbonate is recommended even though it has recently been shown that the calcium ion itself is a stimulant of acid secretion.[7] It is likely that the stimulating effect of the calcium ion is very minor compared to the acid-neutralizing capacity of the calcium carbonate administered.

I prefer to administer calcium carbonate in the form of Tums or Dicarbocil in a dose of 1-2 grams hourly. Grossman recommends 2-4 grams hourly. Others recommend as much as 8 grams hourly. The antacid must be given hourly because the stomach tends to empty at least every hour and probably more frequently. Half-hourly antacids would probably be ideal.

Following two weeks of this treatment a serum calcium level can be checked and, if high, the patient can be placed on one of the non-absorbable alkalies. Those patients who develop hypercalcemia almost always manifest this within two weeks of treatment. In order to be absolutely safe when using calcium-containing antacids, the serum calcium level can be determined every two weeks. The antacid should be given hourly throughout the day and every two hours during the first one or two nights if the patient is having severe

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pain. Following this, the antacid should be given to the patient at night only when he awakens. Intensive, hourly antacid therapy during waking hours should be continued for a minimum of two months in the uncomplicated patient and probably for three months in the patient who has had a complication such as bleeding.[1,8]

If aluminum-containing antacids are used, some of these are poorly reactive with hydrochloric acid and should be avoided.[9] Grossman recommends that following the initial period of treatment no further antacid therapy be given. However, Fordtran[10,11] has published convincing data to show that the traditional administration of an antacid one hour after meals gives prolonged antacid effect and may be beneficial in preventing recurrence of ulcers. Thus, even though Fordtran does not give us control data, I recommend that all ulcer patients take a dose of antacid one hour after meals and before sleep indefinitely.

Anticholinergic drugs are traditionally given for the treatment of duodenal ulcer. These drugs, however, have been shown not to alter the incidence of complications when used as long-term therapy and also may even mask or alter symptoms leading to delay in recognition of recurrence or of complications.[1,5,6] Thus, anticholinergics are not recommended routinely. They should never be used as the sole means of treatment.[1] Their use is contraindicated in the presence of pyloric obstruction, a concomitant gastric ulcer, esophagitis, or in other diseases which contraindicate their use such as glaucoma and prostatic hypertrophy. Thus, anticholinergic drugs are an adjunct to antacid therapy in those patients who fail to respond promptly to antacids and in whom there are no contraindications to the use of anticholinergics. Antropine and belladonna are still as effective as any of the synthetic anticholinergics. For convenience, I prefer to use probanthine in the rare instance when I find it necessary to utilize an anticholinergic. The dose should be individualized so that the patient develops side-effects such as a dry mouth or visual blurring. If the anticholinergics are not given to the point of side-effects there is no effect upon stimulated acid secretion by the stomach.

Sedatives have not been demonstrated to be of value and should not be a routine part of treatment, but if the patient has a problem with anxiety, insomnia or restlessness, sedatives are indicated, and phenobarbital is as effective as any of the more expensive tranquilizers.

Even though the role of diet is commonly discussed in the treatment of duodenal ulcer disease, it plays a minor part. Food is both an antacid and a stimulus for acid secretion. It has been recently shown that proteins, the foodstuffs that have an acid-stimulating effect, also have the ability to neutralize approximately as much acid as they stimulate.[12,13] Fats and sugars do not stimulate acid secretion by a direct effect but do so by distending the stomach. It is true that duodenal ulcer pain is often more likely to follow full-sized meals, particularly after the period of neutralization has given way to a stimulant effect. Thus, some patients do benefit by ingesting six small meals a day rather than three full-sized meals. However, the pattern of food intake and diet cannot be regarded as an important part of the therapy of duodenal ulcer. The lack of effectiveness of rigid dietary control in the treatment of duodenal ulcer is well documented.[14] Thus, I never emphasize dietary therapy to my patients with this disease. The patient can determine the foods that he prefers. If he likes pizza and it causes him no distress, then let him eat pizza. There are a few reasonable interdictions concerning dietary therapy. Caffein is a potent stimulant of acid secretion. Thus, caffein-containing beverages such as coffee, tea and cola drinks and caffein and theobromine-containing foods, such as cocoa, should be avoided. Alcohol is bad because it is not only a powerful stimulant of acid secretion but also a direct mucosal irritant. Acid containing foods should be avoided.[15] The pH of vinegar is 3.1, orange juice 3.4, cranberry juice 2.6, diet cola 2.9, and coke 2.9.[15] A variety of drugs including salicylates and corticosteroids should be avoided, as well as xanthines and reserpine.

It has generally been held that tobacco-smoking is deleterious to the course of duodenal ulcer, but this is not established with certainty.[16,17] If the patient can stop smoking without emotional trauma, this is advised. Nevertheless, if a reduction in cigarette-smoking causes considerable anxiety to a patient, this may have a more harmful than beneficial effect.

General factors such as rest and relaxation should not be neglected. There is evidence to show that bed-rest alone is adequate to heal gastric ulcer, but this has not been tested in patients with duodenal ulcer.[1] Psychotherapy plays no major role in the treatment of these patients although psychological factors are of importance in determining response to treatment.[18] A good doctor-patient relationship will at least make the patient happy even though there is no evidence that it will promote healing of the duodenal ulcer.

In view of the striking frequency of duodenal ulcer, we have little hard data concerning the treatment of this disease. Until we have more control data concerning treatment the outline recommended above is based upon physiological data and seems "reasonable." The paper by Grossman[1] should be read in detail by all who are interested in gaining further insight into the management of duodenal ulcer.

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THE VIEW BOX

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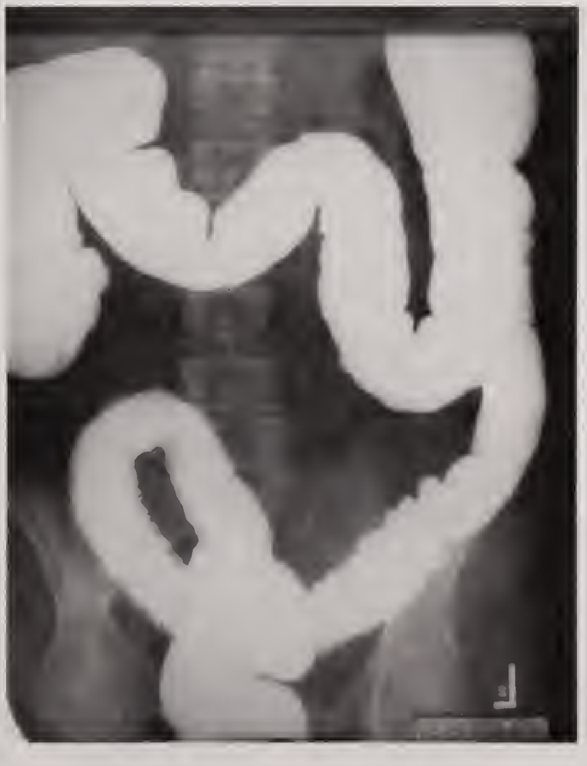

A 60 year old is admitted with a history of bloody diarrhea (5 to 6 movements a day) for several weeks duration. She observed occasional pus with the blood but had no abdominal pain. On external examination there was no abdominal mass, no fistulae or anal ulceration.

(For diagnosis see page 694)

Widespread Brain Damage in Alcoholics*

RAY B. SMITH, PH.D.

Approximately 17% of the more than 5600 patients treated at the District of Columbia's Rehabilitation Center for Alcoholics have been diagnosed as having brain damage. Instruments used for this diagnosis were the Bender Gestalt and the Wechsler Adult Intelligence Scale (WAIS). Most of the men diagnosed as having brain damage had either a long-standing history of brain dysfunction dating back to childhood or had noticeable scars on their head suggesting physical trauma as the agent of the performance loss. Psychometrists at our Center were not able to state that alcohol was a direct factor in such brain damage.

The WAIS measures primarily cognitive functioning and the functioning of other upper brain centers.[1] The Bender Gestalt test measures other aspects of upper brain cognitive and integration processes.[2]

Of greater interest in the present paper is the comment made in the same manual[2] that the Bender Gestalt Test is of little use in measuring brain-loss in alcoholics, the three patients cited being near death at the time the Bender achieved diagnostic significance.

Postmortem neuropathological eximinations of the brains of 70 cases of Korsakoff pyschosis† showed damage in the periventricular and periaqueductal region in every case, with the mammillary body the single most often involved region (95.7% of the cases).[3] The role of the mammillary bodies in learning and memory has been recorded.[5] They appear to lie directly in the pathway of incoming and outgoing information.[6] Clinically the behavior found most frequently in the report cited above[3] was memory impairment. These were evident in 100% of the 70 cases studied. More recently a review of the literature concerning effects of alcohol on memory suggested that "alcohol most severely and selectively disrupts shortterm memory. This disruption is a specific memory deficit common to 'cocktail-party drinking,' alcohol amnesia, and the Wernicke-Korsakoff syndrome, and there is a continuum between the three." [4]

Since alcoholism involves behavior changes in the individual (e.g., tolerance shift), physiological psychologists assume underlying brain changes supporting them. From the studies cited above the area of the brain with the highest probability of change would be the mammillary bodies, and the behavior most likely to be altered as a result would be short-term memory function, one of the known functions of the mammilothalamic projection system.[7] The Benton Visual Retention Test (VRT) was selected to measure this function in three samples of alcoholic patients at our Rehabilitation Center, since it purports to measure shortterm memory functioning.

Method

106 patients were given the Beta I.Q. test and the VRT, one-third each receiving equivalent form C, D, or E. Patients were tested one week after withdrawal and in the order that they arrived at our Center. The I.Q. score was necessary for the interpretation of the VRT.

In a second part of the study 74 patients who had been most free of histories of physical trauma, with no liver involvement, no history of gastritis, no history of delerium tremens or seizures, etc., in other words, our "cleanest" patients physiologically, were tested with the Weschler Adult Intelligence Scale (WAIS) and the Benton VRT.

Finally, 37 patients were selected randomly and given the Beta I.Q. Test and the Benton VRT within a week of withdrawing, then given both tests again an average of 107 days of abstinence at our Center. All Benton VRT's were scored as per the manual[8] using only the Number Correct Score. The Types of Error scores had proved unhelpful in a pilot study with alcoholics.

Participants in the three studies were all males and had ages averaging 43.5 years, with approximately a 50/50 White-Negro ratio of participants. All received administration A of the Benton.

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† Korsakoff psychosis is one of the most frequent brain-damage syndromes among alcoholics.

Results

Table 1 shows the distribution of scores obtained by the three groups of patients, as compared with the scores that would be expected from their intelligence test scores and ages. Based on original Benton VRT test norms, the distribution should be normal around a difference score of zero. Instead, every group of alcoholics yielded a markedly skewed distribution to the right, i.e., in the brain damage direction of the scale. A summary of this distribution is found in table 2, in which 86.6 % of the men failed to make the score expected. When compared with the Benton VRT Manual impairment categories, at least 70.5 % of the alcoholics are in the questionable or worse range. The Manual's impairment categories are based on a normal distribution of scores, however, and if in the present sample one substracts the number who were one or more over their expected score from those down one or more from their expected scores, the chance variation around the sample mean can be roughly estimated. That yields roughly 83.8 % of the present sample with a manifest performance deficit.

Table 3 gives the comparative performance by the group who underwent re-testing after an average of 107 days.† Statistically, there was no change in overall performance during this length of sobriety. The second section of the table shows that there was almost as much loss as there was gain in the overall raw score during the period. Equally interesting is that the Beta I.Q. changed, averaging a gain of 6.5 I.Q. points, putting 40.5 % of the group into a different intelligence category after 107 days of sobriety than the one in which they were placed shortly after withdrawing from their last drinking episode (all higher except one). Nonetheless, their overall Benton VRT performance remained substantially the same.

Discussion

Alcohol appears to yield loss in the lower brain areas, as found in postmortem examinations and as clinically verified. Now the Benton Visual Retention Test selectively screens out those who have suffered short-term memory loss but little or no damage to the upper integration areas of the brain as measured by the WAIS or

TABLE 1: Performance on the Benton Visual Retention Test By Three Groups of Alcoholics

	Difference Between Expected and Obtained Scores									
Group	+3	+2	+1	0	−1	−2	−3	−4	−5	−6 plus
Patients tested on entering Rehabilitation Center (Beta I.Q.) (106)	0.9%	0.9%	1.9%	4.7%	17.9%	16.0%	18.9%	15.1%	18.9%	4.8%
Patients without previous medical pathology (WAIS I.Q.) (74)	0.0	0.0	1.4	14.9	12.1	18.9	17.6	12.1	14.9	8.1
Random patients (Beta I.Q.) (37)										
Test:	0.0	0.0	2.7	18.9	19.0	35.1	10.8	10.8	0.0	2.7
Re-test:	0.0	2.8	2.8	25.0	19.4	19.4	11.1	13.9	2.8	2.8
Total 217*	0.5	0.5	1.8	10.6	16.1	20.3	17.0	13.4	14.3	5.5

* Total Performance is based on the initial testing of the random patients group.

TABLE 2: Summary of Performance on the Benton Visual Retention Test By Alcoholics in the Three Groups

Group	Obtained Expected Score Or Better	Obtained Less Than Expected Score	Obtained Two Points or More, Less Than The Expected Score*
Patients tested on entering Center	8.4%	91.6%	73.7%
Patients without previous medical pathology	16.3	83.7	71.6
Randomly selected			
Patients Test:	21.6	78.4	59.4
Re-test:	30.6	69.4	50.0
Total 217†	13.4	86.6	70.5

* According to the Benton VRT Manual, persons with acquired impairment would fall into this category.

† Based on first testing of randomly selected patients group.

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† The author is indebted to Eric Weingarten, Ph.D., and his staff for obtaining these scores.

TABLE 3: Comparative Performance By The Test Re-test Group

Testing	Above Expected Score	Made Score Expected	Below Expected Score
First (37)	2.7%	18.9%	78.4%
Second (36)*	5.6	25.0	69.4
	Score Correct, Up	Score Correct, No Change	Score Correct, Down
First vs. second (36)†	44.0%‡	25.0%	31.0%§

* These expected scores were based on the re-test Beta I.Q., 40.5% of which went up a mean of 6.5 I.Q. points.

† These are raw scores correct without considering I.Q. or expected scores.

‡ Average gain 1.6 points.

§ Average loss 1.9 points.

Bender Gestalt. Insofar as such memory impairment is diagnostic of Korsakoff degeneration, there should be a relationship between the amount of loss on the Benton and the likelihood of a diagnosis of Korsakoff. That such a relationship exists has been observed repeatedly in our Center's diagnostic section.

Cases of Korsakoff present a special problem in a rehabilitation center which stresses individual and group psychotherapy, in that persons with such memory impairment do not integrate new learning experiences into their pre-Korsakoff personality.[3] We have observed that persons with increasingly more profound loss on the Benton test are increasingly less likely to show improvement in psychotherapy or benefit from our educational therapy programs. Attempts at vocational rehabilitation have been noticeably unsuccessful with patients who score 50% or below their expected score on the Benton Test. Patients diagnosed as having Korsakoff pychosis by psychiatrists at our Center have uniformly scored 50 to 75% below their expected Benton Test performance, and occasionally a score of zero is obtained.

The Benton Visual Retention Test may be helpful in screening for the most suitable candidates for the various therapy programs in a crowded rehabilitation center, especially those therapies involving personality changes and the integration into the personality of new learning experiences. It takes from five to 10 minutes to administer (administration A), and most of the personality characteristics observable on the Bender Test can be obtained from the Benton Test by a trained clinician.

The present study suggests that by the time an alcoholic reaches a public rehabilitation center there is something like an 80% chance that he is suffering brain damage sufficiently advanced to be measurable with the Benton VRT, while 60% of these may escape detection on the WAIS or Bender Gestalt. Therefore, the Benton Test is recommended as a first filter test for use in alcoholism treatment centers.

Finally, from a theoretical point of view the second part of the study in which a majority of otherwise healthy patients were shown to have a short-term memory performance deficit is most interesting. It raises the question of other brain changes that yield loss of control over drinking, increased tolerance, etc. Such control centers, like the mammillary bodies, lie against ventricle III in the brain or near the only capillaries of the blood-brain barrier outside of the choroid plexus to have fenestrations. This suggests that alcohol per se may not be the villian in the behavioral changes defining alcoholism but perhaps a breach of the blood-brain barrier by secondary toxins in this area of the brain. Further studies are being pursued in our laboratories on this question.

Summary

Over 70% of the patients tested at the District of Columbia's Rehabilitation Center for Alcoholics were found to have short-term memory deficits as tested by the Benton Visual Retention Test for brain damage. It is inferred that the mammillary bodies at the base of the hypothalamus are probably the area of lesion. This gives rise to a new theory of alcoholism in which the blood-brain barrier, which is fenestrated in this area, is breached by toxins other than alcohol which cause underlying brain changes and consequent shifts in behavior toward that defining alcoholism.

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Laurence-Moon Syndrome: Evaluation of Endocrinological Function and Phenotypic Concordance and Report of Cases*

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The Laurence-Moon syndrome is a hereditary disorder characterized by structural and functional abnormalities of organs and tissues of diverse embryonic derivation. The five principal features are polydactyly, pigmentary degeneration of the retina, obesity, oligophrenia, and hypogonadism.[1-3] Various combinations of these abnormalities have been described in several hundred families.[4-6] Some of the affected individuals may also have brachycephaly, short stature, congenital heart disease, deafness, and various neurological disorders.

We have studied four siblings from two families representative of the spectrum of this syndrome, paying particular attention to gonadal function and pituitary-gonadal axis. In addition, the extent of phenotypic concordance among the affected siblings was assessed on compiled data from case reports published between 1960 and 1970, including our own patients.

Report of Cases

Case 1. A. W. is a 22-year-old white man (Fig. 1A). The birth weight was 4.7 Kg following an uncomplicated 40-week gestation. Obesity has persisted since birth. Extra toes on each foot were removed at the age of nine months. Early motor and intellectual developments were normal. Visual loss began at the age of eight years, and the patient entered a school for the blind at the age of 16 years. Pubertal changes began at the age of 17 years. He is unmarried, reads braille, and helps in running the family farm.

Both parents are of above average height and are in good health. The mother and many of her relatives are obese. There was no family history of consanguinity, blindness, polydactyly, mental retardation, or hypogenitalism. Their first offspring, a 26-year-old woman, is moderately obese but otherwise normal, the second, a male, died in infancy of congenital heart disease. The third and fourth offspring are the affected brothers (A. W. and B. W.).

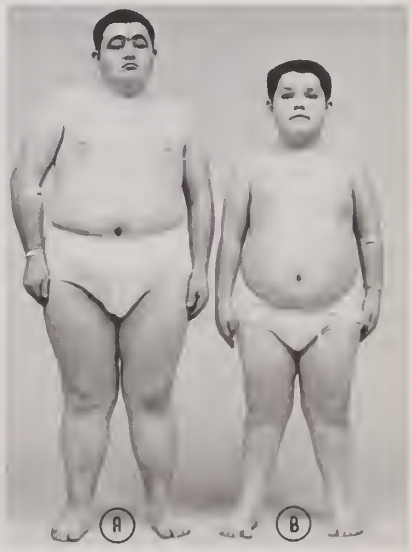

FIG. 1. Affected brothers with Laurence-Moon syndrome. A–A. W. (Case 1), B–B. W. (Case 2).

The patients height was 161 cm (below the third percentile), the weight 99 Kg (above 97th percentile), the head circumference 59 cm (above 97th percentile), and the cephalic index 0.82. Vision was limited to the perception of strong light. The retinal arteries were very thin, and particles of black pigment were seen in the diffusely degenerated retinae (Fig. 2A). An electroretinogram showed complete absence of responses. The I.Q. was 88 (Wechsler-Bellevue). Examinations of respiratory, cardiovascular and nervous systems were normal. Beard, axillary and pubic hair showed an adult male pattern. The testes were descended and measured 2.3 x 2.1 x 4.3 cm bilaterally, with an estimated volume of 10.2 ml. The penis was 12 cm in length.

Urinalysis, hemogram, serum proteins, cholesterol, electrolytes, blood urea nitrogen, and glucose tolerance test were within normal limits. Lymphocyte karyotype was 46 XY.

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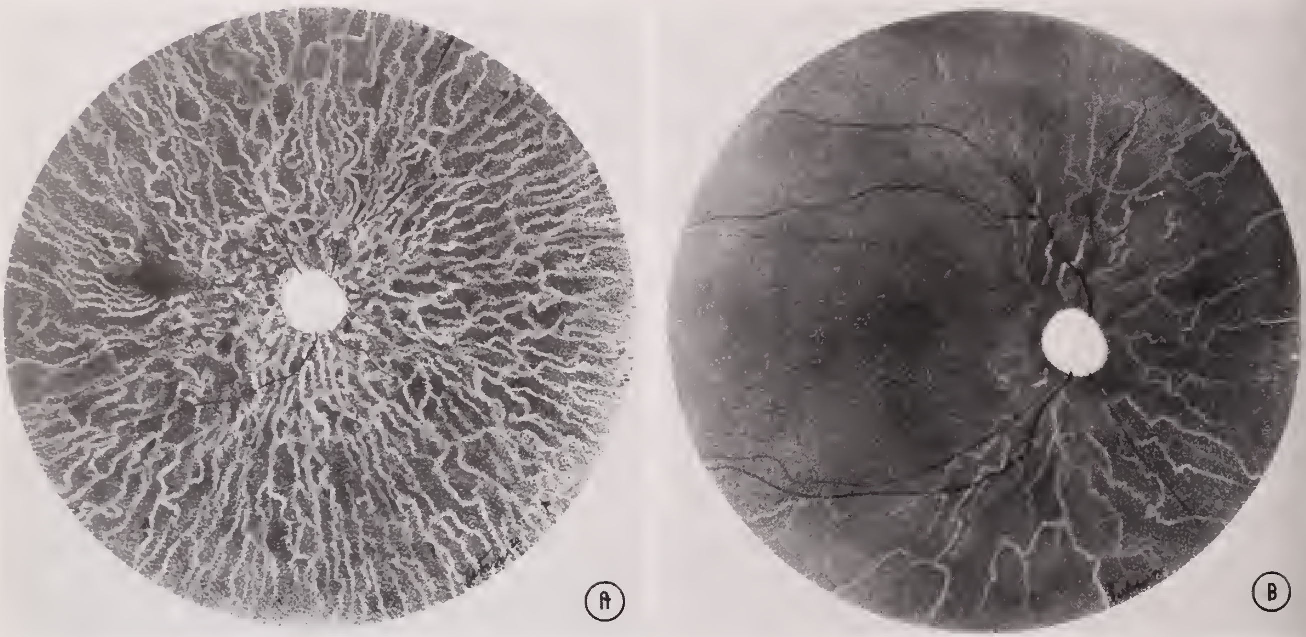

FIG. 2. Drawings of optic fundus showing pigmentary degeneration of retina: A–fundus of patient A. W. (Case 1); B–fundus of patient B. W. (Case 2).

EEG and EKG were normal. X-ray films of the skull, chest, spine and extremities showed a mild thoracic scoliosis, short phalanges and metacarpal bones, and evidence of amputated supernumerary toes. An intravenous pyelogram revealed terminal dilatation and rounding of the calyces. An ejaculate showed a volume of 3.0 ml and 72 million spermatozoa per ml with normal motility and morphology. Further endocrine studies are given in table 1.

Case 2, B. W. is a 9-5/12-year-old brother of Case 1 (Fig. 1B). The birth weight was 4.5 Kg following an uncomplicated 40-week gestation. Obesity has persisted since birth. An extra finger on each hand and an extra toe on each foot were removed at the age of nine months. Early motor and intellectual developments were normal. Vision began to fail at the age of eight years, but the boy continued to do well in a regular school.

The height is 142 cm (65th percentile), the weight 64 Kg (above 97th

TABLE 1: Values of Various Endocrine Tests in the Four Patients Studied

	Age Sex	PBI	T4	Total Urinary Gonadotropins	Urinary Steroids mg/24 hr.		Plasma T. ng/100 ml		Plasma G.H. ng/ml	
Case No.	Yrs.	µg/100 ml		MU/24 hr.	17-OH	17-Keto	B	HCG	B	ATT
1 (A. W.)	22/M	5.7	5.4	>10 (3) >50 (2)	22	25	310	583	1.0	22
2 (B. W.)	9/M	8.3	7.4	>10 (4) <10 (1)	7	11	74	284	1.4	4.5
3 (S. M.)	28/F	7.1	5.6	<10 (2)	5	14	–	–	–	–
4 (N. M.)	20/F	6.8	5.2	<10 (2)	8	12	–	–	–	–
Normal value:	Adult	4-8	3.1-6.7	50-200	M5-23 F3-15	M9-22 F6-15	400-1000		1.0	increase of >5 ng/ml
	Children	4-8	3.1-6.7	3			<100		1.0	

B, Basal values; HCG, following stimulation with human chorionic gonadotropins; G.H., Growth hormone; T, testosterone; ATT, arginine tolerance test.

percentile), the head circumference 5.75 cm (above 97th percentile), and the cephalic index 0.83. Peripheral vision was constricted and central vision was 20/100. The retinal arteries were narrow, and degenerative changes were present together with scattered black pigmentation (Fig. 2B). An electroretinogram showed complete absence of responses. Verbal I.Q. (Wechsler-Bellevue) was 100. The respiratory and cardiovascular systems were normal. Mild degrees of genu valgum and pes cavus were present. Hearing and neurological examinations were normal. Pubic hair was absent. Both testes were descended and measured 0.5 x 0.5 x 1.0 cm. The penis was 4 cm in length.

Urinalysis, hemogram, blood chemistries, and glucose tolerance test were normal. No abnormalities could be found on X-ray examinations of the skull, chest, spine and extremities. An intravenous pyelogram showed terminal dilatation and rounding of the calyces together with distortion of the collecting system in the lower pole of the right kidney (Fig. 3). The presence of a mass was confirmed by renal arteriography, and an exploratory operation was performed. A simple cyst was found and unroofed. Results of endocrine tests are shown in table 1.

Family M. The mother is obese and 157 cm tall. She has maturity-onset diabetes, non-toxic goitre, chronic lymphocytic leukemia, and rheumatic heart disease. Her two siblings are normal. The father is 170 cm tall and is unable to smell. His mother became demented in her early fifties; her brother's son was blind and died at the age of 27 years. No family history of consanguinity or polydactyly was elicited. There were two children, both affected in the last generation.

Case 3. S. M. is a 28-year-old woman (Fig. 4A). The birth weight was 2.3 Kg; the term pregnancy was complicated by first trimester spotting. She stood without support at 19 months, walked at 24 months, and spoke at three years. Impaired vision was first evident at two years of age. Attacks of rheumatic fever with carditis occurred at the ages of eight and 15 years. She also had recurrent urinary-tract infections, and an intravenous pyelogram at the age of 11 years showed a vesical diverticulum. Menses began at the age of 14-8/12 years and continued irregularly until bilateral oöphorectomy and subtotal hysterectomy were performed at the age of 20 for ovarian cysts. Examination at the age of 15-4/12 years disclosed tubular vision with central acuity of 20/100. A "bone corpuscles" type of pigmentary deposit was noted at the periphery of the retina. An electroretinogram revealed complete extinction of potentials. On Stanford-Binet score the I.Q. was 30. Re-examination at the age of 28 years showed the height to be 144 cm (below the third percentile), the weight 60.7 Kg (50th percentile), the head circumference 51 cm (third percentile), and the cephalic index 0.87. She was slightly obese and had marked tibial edema. Cardiac examination suggested aortic and mitral insufficiency. Severe kyphoscoliosis was present. She showed hyperextensibility of the joints, long, tapering fingers, and hyperreflexia. The breasts were fully developed, and the body hair was in an adult female distribution. Thery was a normally developed vaginal introitus, unstimulated vaginal mucosa, a mature cervix, and no palpable ovaries or uterine fundus.

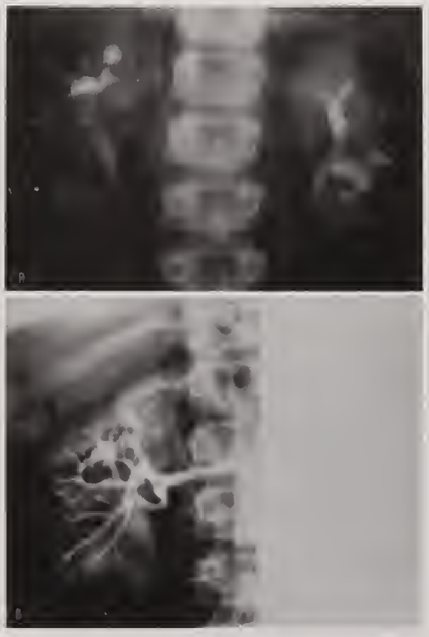

FIG. 3. Intravenous pyelogram and renal angiogram of patient B. W. (Case 2), showing dilatation and rounding of terminal calices.

Case 4. N. M. is a 20-year-old sister of Case 3 (Fig. 4B). Her birth weight was 2.7 Kg.; a full-term pregnancy was complicated by congestive heart failure in the last trimester. Hypotonia was evident from birth. She first sat without support at three years and stood and spoke at four years. At the time she had visual difficulties, manifested by bumping into stationary objects. Funduscopic examination at 7-8/12 years showed narrowing of the retinal arteries, peripheral depigmentation, and the presence of a few particles of black pigment. The electroretinogram showed absence of responses. She scored an I.Q. of 32 on the Stanford-Binet scale. Menses began at the age of 15 years, and she has had irregular one to three-month cycles since that time. Attacks of rheumatic fever with carditis occurred at the ages of 13 and 16. At the age 20 she was without peripheral edema, the height was 137 cm (below the third percentile), the weight 65 Kg. (third percentile), the head circumference 50 cm (below the third percentile), and the cephalic index 0.83. Vision was just adequate to recognize faces. Cardiac examination was consistent with mitral stenosis. She had hypotonia, severe kyphosis, sloping shoulders, long, tapering fingers, and hyperreflexia. Dark, coarse hair was present on the thighs and upper lip, but the pubic hair was in normal triangular distribution. Pelvic examination showed normal clitoris and vaginal introitus, estro-

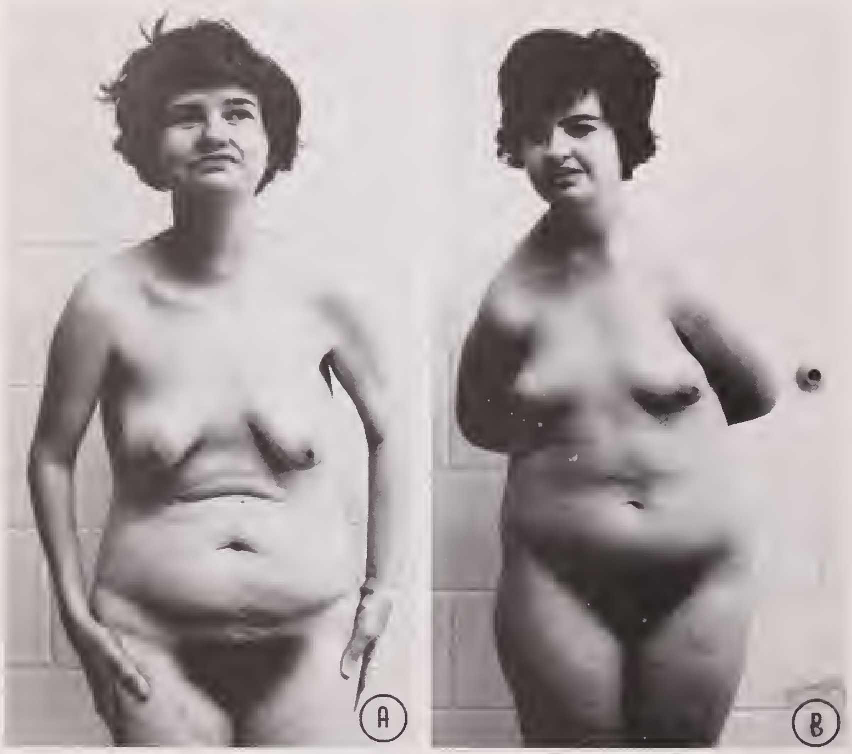

FIG. 4. Affected sisters with Laurence-Moon syndrome. A–S. M. (Case 3), B–N. M. (Case 4).

genized vaginal mucosa, blood-tinged cervical mucus, and normal adult ovaries and uterus.

Urinalysis, complete blood count, calcium, phosphorus, creatinine, cholesterol, and glucose were normal. X-ray films of the chest, skull, and skeleton revealed normal findings. Endocrine studies are shown in table 1.

Results

Following a variable delay, all three adult patients attained mature gonadal function, and the findings in the nine year-old boy (Case 2) were consistent with the normal prepuberal male. The affected brothers in the W. family had three out of five main features of the Laurence-Moon syndrome: polydactyly, retinitis pigmentosa and obesity, while the affected sisters of the M. family had retinitis pigmentosa, oligophrenia, a moderate degree of obesity, but no polydactyly.

Table 1 shows the results of various endocrinological tests performed on these patients. Total urinary gonadotropins in patient A. W. (Case 1) were in the post-puberal range. His plasma testosterone was in the low-normal, adult-male range and nearly doubled following stimulation with human chorionic gonadotropin. The normal sperm count provided conclusive evidence of adult sexual development. The gonadotropin excretion by his nine year-old brother was greater than 10 mouse-uterine units per 24 hours in four specimens, clearly separating him from pubertal patients with hypogonadotrophic hypogonadism. Furthermore, his plasma testosterone values more than doubled following human corionic gonadotropin. We expect that he will resemble his brother in experiencing complete sexual maturation. Our female patients (Cases 3 and 4) had menarche at somewhat later than the mean age of 13 years and had irregular menses thereafter; their urinary steroids were within normal range for adult females. While failure to detect gonadotropins in 24-hour urine specimens from normal women occurs about 30% of the time in our laboratory, the same cannot be said for castrate women. Thus the observation of no gonadotropin in two specimens from patient S. M. (Case 3) was unexpected, and we are unable to explain this finding. Diabetes insipidus was excluded clinically and by the demonstration of a rise in the urine osmolality to a level twice the plasma osmolality following overnight water deprivation. Protein-bound iodine and serum thyroxine values were normal. Urinary excretion of 17-hydroxysteroids and 17-ketosteroids was in the normal range. Patient A. W. (Case 1) showed a normal growth hormone response to arginine infusion, and his younger brother's (Case 2) response was somewhat blunted; the significance of this should have been determined by repeating the test after estrogen priming. The values of the insulin hypoglycemia test and Metyrapone test fell within normal limits. Thus, we found little evidence of dysfunction of target glands, pituitary, or hypothalamic-pituitary-gonadal axis.

Table 2 shows the distribution of clinical findings in 59 patients compiled from published case reports between 1960 and 1970.[7-24] Only those patients were accepted for tabulation whose case histories were judged to contain enough information regarding principal clinical findings. Thirty-one patients (52.4%) were under 15 years of age with a median of seven years and four months, and 28 patients (47.6%) were over 15 years old with the median age of 22 years and five months. Since 52.4% of all patients were under the age of 15 years, the possibility of delayed puberty

TABLE 2: Incidence of Various Abnormalities in the Laurence-Moon Syndrome in Patients as Assessed During Examination Under 15 Years of Age and 15 Years and Older

						Gonadal Function				Congenital Abnorm.		
Patients	No. of Cases	Polydactyly	Obesity	Subnormal Mentality	Imp. Vision & Ret. Pigm.	Hypog.	Normal	Too Young for Assessm.	Stature Subnormal	Urinary Tr. & Genital Malformation	Visceral & Heart	Chronic Urinary Disease
Under 15 years	31	22 (71%)	25 (80.6%) (1?)	24 (77.4%) (3?)	24 (77.4%) (2?)	?	6 (19.3%)	25 (80.6%)	7 (22.5%) (4?)	7	2	2
15 years and over	28	17 (60.7%)	19 (67.8%) (1?)	19 (67.8%) (1?)	26 (92.8%)	14 (50%)	12 (42.8%)	2 (7.1%)	24 (60%) (2?)	4	4	2
Total	59	39 (66.1%)	44 (74.5%)	43 (72.8%)	50 (84.7%)	?	18 (30.5%)	27 (45.7%)	21 (45.7%)	11 (18.6%)	6 (10.2%)	4 (6.8%)

forestalls definitive assessment of gonadal function in the majority of these subjects. Of the whole series of 59 patients, only 14 or 23.7% were definitely hypogonadal; gonadal function was judged satisfactory in 30.5% because of the presence of menstruation or morphologically normal sperm, or normal external genitalia and gonads, but in 45.8% the subjects were too young for definitive assessment. Obesity (as interpreted by us from standard weight tables) was present in 74.5%, with a slightly greater incidence in those under 15 years of age. Polydactyly was present in 66.1% and mental subnormality in 72.8%. Impairment of vision and retinitis pigmentosa were more common in subjects over 15 years of age (92.8%) than in those under 15 years of age (79.6%). This is understandable, since retinal lesions generally develop past the age of four to 10 years. Subnormal height (as interpreted by us from the stature tables) was encountered in 35.5% of the total series. In 28.8% of all patients either genitourinary, skeletal, visceral, or heart malformations were present. Chronic renal disease, usually pyelonephritis or uremia, occurred in 6.8% of all cases. This is probably an underestimate because of the predominantly young age of the subjects in the sample and omission of such tests as intravenous pyelogram, especially in patients already receiving institutional care. Our finding of blunted calices in both young siblings may indicate that this structural anomaly could be present in many patients if specifically looked for.

We have considered it important to assess the extent of phenotypic concordance of these principal abnormalities among the affected siblings in different families. As it may be seen in table 3, there is a very striking intra-family concordance of the abnormalities while marked inter-family differences exist. Thus, either complete or near complete concordance was present in all 13 families with two or more affected siblings.

Discussion

The results of the present investigation underscore two important aspects of the Laurence-Moon syndrome: the status of hypogonadism and the unexpectedly high intra-family concordance of the principal clinical abnormalities in the presence of considerable inter-family differences.

The overall frequency of hypogonadism in males was estimated at 83% by Bell[5] in her extensive review and by Klein and Ammann[6] at 85.7%. These figures include boys with undescended testes or a phallus which appears small because of abnormal obesity or delayed adolescence. The same authors quoted the incidence of primary amenorrhea and/or deficient breast development in females at about 50%. However, it is evident from the compiled published case reports between 1960 and 1970 (table 2) that slightly more than one-half of patients (31 out of 59) were under the age of 15 years when examined and that definitive gonadal function could not be assessed accurately if the possibility of delayed puberty is considered. Among 28 persons examined when they were 15 years of age or older, hypogonadism was thought to be present in 50% of cases, a value substantially lower than the average for combined sexes quoted in the literature. These observations suggest that the delay in sexual development occurs frequently among these patients for reasons that are presently unclear.

The type of hypogonadism found in the few adult males who have been adequately studied has varied. Both hypogonadotrophic hypogonadism and primary testicular failure have been described.[14,15] Roth et al[25] reported men with Laurence-Moon syndrome who had evidence of spermatogenesis on testicular biopsy, and one of our patients (Case 1) had the usual number of well-formed spermatozoa in the ejaculate. Clinical signs of delayed sexual development or hypogonadism are among the more obvious indices of endocrinopathy. Studies of endocrine function (thyroid, adrenal, pituitary) are generally normal, and at least half the patients when examined during adult years have normal or near normal functioning gonads. We have tested the hypothalamo-pituitary axis in two patients (Cases 1 and 2) and found no obvious abnormality.

The second question relates to the high concordance of clinical abnormalities among the siblings of the same families. This is clearly demonstrated in table 3, but the explanation is not at hand. During the past decade an unprecedented progress has been made

TABLE 3: Assessment of Phenotypical Concordance Among Siblings of Laurence-Moon Syndrome

Family & Sib. Identification	Sex/Age y.	Polydact.	Obesity	Ret. Pigm.	Mental Subnorm.	Hypogonad.
16 Ba	F/50	+	+	+	+	–
Bb	F/33	+	+	+	+	+
Bc	M/24	+	+	+	+	+
Bd	M/15	+	+	+	+	+
9 Aa	F/48	–	–	+	+	+
Ab	M/42	–	–	+	+	+
Ac	M/39	–	–	+	+	–
Ad	F/37	–	–	+	+	+
Ae	F/34	–	–	+	+	+
7 Aa	M/5	+	+	–	+	*
Ab	M/1	+	+	–	+	*
Ac	M/2 d	+	+	–	*	*
11 Aa	F/27	+	+	+	+	–
Ab	F/17	–	+	+	+	–
Ac	M/17	+	+	+	+	–
8 Aa	M/6	+	+	+	+	*
Ab	M/9	+	+	+	+	*
15 Aa	M/21	–	–	+	+	+
Ab	F/23	–	–	+	+	+
16 Aa	F/30	+	+	+	–	–
Ab	F/35	+	+	+	+	–
25 Aa	M/6	+	+	+	+	*
Ab	M/4	+	+	*	+	*
18 Aa	F/14	–	+	+	+	–
Ab	M/13	–	+	+	+	–
20 Aa	M/13	–	+	+	+	*
Ab	F/12	–	+	+	+	*
23 Aa	F/14	+	+	+	+	*
Ab	M/1	+	+	*	*	*
Family W Case 1	M/22	+	+	+	–	–
Case 2	M/9	+	+	+	–	–
Family M Case 3	F/28	–	+	+	+	–
Case 4	F/20		+	+	+	–

* Patient too young for assessment of gonadal function, oligophremia, or retinitis pigmentosa.

in the elucidation of sequences of molecular events in many inborn errors of metabolism.[26,27] In a number of disorders we can now trace consecutive metabolic steps from the mutant gene through the deficiency in the corresponding enzyme, to the overt clinical manifestations of the disturbed body metabolism.

By contrast, little is known about genetic mechanisms which govern structural differentiation during development or maintanence of the intactness of the organs and tissues during mature life. In this connection the presence of true malformations as well as asynchronous appearance of the principal manifestations during life of the patients with Laurence-Moon syndrome is particularly interesting. The teratogenic period for polydactyly is between five and seven weeks of gestation and of other skeletal and visceral malformations a few weeks later. If present, retardation of mental and motor development becomes evident during early infancy. Obesity in Laurence-Moon syndrome mainfests itself in the early years of life and at times is present at birth, as in our Cases 1 and 2. Retinal degeneration leading to blindness appears usually between 4 and 10 years of age, while clinical signs of delayed sexual development or frank hypogonadism may not be apparent until late teenage years.

Our finding of a high phenotypic intra-family concordance (table 3) while the inter-family differences in grouping of the five principal abnormalities are marked, strongly argues against random occurrence of such combinations. Among plausible genetic explanations are the following: a) presence of multiple alleles – different alleles being affected in different families; b) presence of nonhomologous gene with related but different primary effects; and c) existence of only one mutant allele in one locus but the expression of the syndrome modified by other genes and environmental circumstances. In this particular syndrome, multifactor inheritance appears to be less likely. Also, it must be considered that our data cannot exclude the possibility of a particularly unstable area on a chromosome which is readily involved in such chromosomal aberrations as inversion, deletion or duplication of a variable length of the segment. Harnden[28] and Leon[29] have made a brief reference to some of these possibilities while discussing general principles of inheritance of complex disorders.

Summary

Clinical features of the Laurence-Moon syndrome are suggestive of endocrinopathy combined with frequent mental retardation and blindness. We have studied affected siblings in two representative families with the Laurence-Moon syndrome in whom sexual maturation, though delayed, was ultimately completed. Our data and the results of the review of the recent literature have led us to propose that delayed puberty is a frequent occurrence among these patients, leading to a falsely high estimate of the incidence of hypogonadism in this syndrome.

A new feature became evident from analyzing the tabulated data in 13 families with multiple sibling involvement. There is a very striking intra-family concordance of the principal abnormalities while marked inter-family differences exist. Of the possible explanations of the genetic mechanism responsible for this variability of phenotypic expression, the following have been discussed: 1) presence of multiple alleles, a different one being affected in different families, 2) the presence of nonhomologous gene with related but different primary effect, and 3) the existence of only one mutant allele in one locus but the expression of the syndrome modified by other genes and environmental circumstances.

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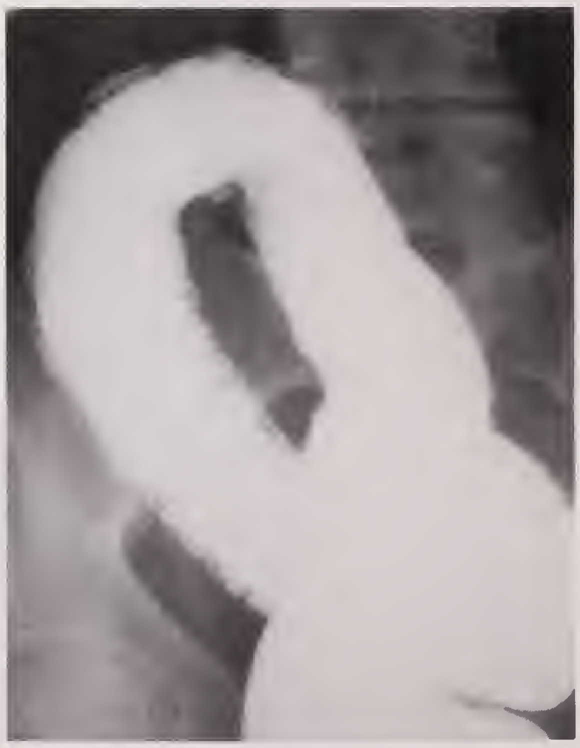
THE VIEW BOX

(from page 683)

DIAGNOSIS: Ulcerative colitis



Note in the Barium Enema the involvement of rectum (involved in about 95% of cases) and sigmoid incontinuity. There is a jagged contour with superficial and undermining mucosal ulcerations. The remainder of the colon and terminal ileum was normal. A rectal biopsy showed changes of ulcerative colitis.

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Current Aspects of Digitalis Therapy*†

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In recent years substantial information regarding the mechanisms of action of the digitalis glycosides has accumulated. It is now generally accepted that the beneficial effects of digitalis in patients with congestive cardiac failure result from direct stimulation of the contractile state of the myocardium. Thus, in hemodynamic studies on patients exhibiting chronic heart failure of the low-output type, it has been shown that digitalis augments the cardiac output and lowers the abnormally elevated left ventricular end-diastolic pressure. It has also been demonstrated that, even though cardiac glycosides exert little effect on the pumping action of the normal ventricle as expressed in the cardiac output, they do exert a stimulating influence on normal cardiac muscle.

Myocardial Oxygen Balance

Among the major determinants of cardiac performance is the balance between oxygen supply and demand. Myocardial oxygen consumption (MVO_2) depends upon a number of factors, the most important of which are heart rate, wall-tension (which is directly related to intraventricular pressure and heart size), and the contractile state of the myocardium. It appears that the increase in contractility produced by digitalis augments MVO_2. However, reduction in heart rate caused by digitalis tends to have the opposite effect. In the dilated, failing heart, which becomes smaller after glycoside administration, the concurrent reduction in wall-tension counteracts the oxygen cost of augmented contractility. Thus the effects of digitalis on MVO_2 depend on the state of compensation existing at the time the drug is administered.

Vascular Effects

The effects of digitalis on flow and resistance in various vascular beds have only recently been determined in the conscious dog. After the animals' full recovery from the implantation of Doppler flow-probes on the aorta and left circumflex coronary, it was found that ouabain caused coronary vasoconstriction despite the increase in MVO_2 which would have been expected to produce dilatation in the coronary vascular bed. Whether the results in normal dogs are applicable to patients with coronary vascular disease remains to be demonstrated. In normal conscious dogs with chronically implanted flowmeters, digitalis causes constriction in the renal and iliac beds but dilatation in the mesenteric bed. In contrast, in dogs with heart failure, flow increased in all beds studied and vasodilatation occurred in response to ouabain, presumably as a consequence of increasing cardiac output, thereby reducing the reflex vasoconstriction which characterizes the low-output state.

Myocardial Infarction

Although digitalis is unequivocally indicated for left ventricular failure complicating chronic valvular and coronary artery disease, the available experimental and clinical data still leave many unanswered questions concerning the role of digitalis therapy after acute myocardial infarction. It is postulated that the fate of the peri-infarction zone, with the limited oxygen available to it, is influenced profoundly by the oxygen requirements of the tissue. Increasing the latter in the non-failing heart of dogs with experimental myocardial infarction by means of ouabain and other positive inotropic stimuli intensifies ischemia and ultimately results in more extensive infarction. In contrast, reducing myocardial oxygen demands by administering digitalis to the infarcted heart which is failing may permit survival of the underperfused zone bordering the infarct.

Thus it seems clear that little is to be gained by glycoside administration to patients with uncomplicated infarction who do not have cardiomegaly. The role of digitalis therapy in the treatment of cardiogenic shock remains undefined, but, based on the data available, its efficacy in this syndrome must be questioned. Digitalis appears to be indicated for the treatment of rapid atrial fibrillation complicating acute myocardial infarction. Other supraventricular arrhythmias, such as atrial flutter and ectopic atrial tachycardia, frequently require larger doses of the glycoside and other means of therapy, such as anti-arrhythmic agents or electrical

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conversion, may be preferable in such cases. Digitalis is commonly utilized early in the treatment of congestive heart failure and cardiomegaly complicating acute myocardial infarction despite the paucity of experimental and clinical evidence in support of such therapy. Whether the digitalis glycosides should continue to be used routinely in such patients remains a subject for continuing clinical investigation.

Pharmacology and Assay

The metabolic half-times and excretion rates of short, intermediate, and long-acting glycosides have been well characterized. Renal insufficiency as well as old age are associated with impaired renal excretion of digitalis glycosides, suggesting that digitalis dosage should be modified in such patients.

More recently, radioimmunoassay for serum digoxin concentration has come into routine clinical use in a number of centers. Serum digoxin concentrations exceeding two nanograms (ng)/ml appear to be associated with an increased frequency of toxic manifestations. Smith and Haber reported that patients with supraventricular tachycardia and block apparently induced by digitalis, and those with ventricular dysrhythmias had mean serum digoxin concentrations of 4.1 and 3.7 ng/ml, respectively. In contrast, patients with atrial fibrillation who had a reduced ventricular response and ventricular premature beats, and patients with sinus rhythm and second or third degree atrioventricular block, had levels of 2.2 and 3.2 ng/ml, respectively. Overlap may occur, and patients with levels considerably greater than 2.0 ng/ml may have no symptoms, while those with lower levels (but usually not less than 1.6 ng/ml) may demonstrate electrocardiographic abnormalities, especially ventricular premature beats. Nevertheless, in patients whose serum digoxin levels exceed 2 ng/ml, it is prudent to discontinue the glycoside until the serum level falls below this concentration. With the availability of this radioimmunoassay technique, it should be possible to analyze critically digitalis dosage schedules in relation to evidence of both beneficial effects as well as toxicity in patients with acute myocardial infarction and to determine definitively whether acute myocardial infarction sensitizes patients to digitalis-induced arrhythmias.

Although our knowledge of the mechanisms of action and clinical utilization of this most useful of cardiac drugs has advanced considerably in the past decade, much work remains. Directions for future basic research include clarification of the digitalis receptor and definition of the subcellular mechanism of glycoside action. On a clinical level the use of digitalis in acute myocardial infarction in man remains an important topic for future investigation.

Gallop Rhythm*

ROBERT A. O'ROURKE, M.D.†

The term "gallop rhythm" was originated by Professor Bouillaud and propagated by his pupil Potain over a century ago. However, even today the majority of gallop sounds are unrecognized or misinterpreted. This is unfortunate because gallop rhythm is frequently the only positive physical finding in patients with heart disease, and its presence often has important diagnostic and therapeutic implications.

Gallop rhythm is an auscultatory phenomenon in which a tripling or quadrupling of heart sounds resembles the canter of a horse. Tachycardia need not be present. Gallop sounds are low-frequency diastolic events related to two periods of ventricular filling—the rapid filling phase (third heart sound, ventricular gallop) and the presystolic filling associated with atrial systole (fourth heart sound, atrial gallop). Both third and fourth heart sounds may be present in the same patient. During tachycardia or advanced first degree A-V block, both gallop sounds may occur at almost the identical time, producing a summation gallop. The summation gallop may be confused with the diastolic rumble of mitral stenosis. However, decreasing the heart rate by transient carotid-sinus pressure will separate the two gallops and distinguish them from a diastolic rumble.

Fourth Heart Sound

The fourth heart sound (presystolic gallop, atrial gallop) is a low-frequency sound produced in the ventricle during the ventricular filling associated with an effective atrial contraction. The atrial gallop is occasionally heard in patients with no evidence of heart disease, particularly during times of high cardiac output such as occurs with thyrotoxicosis or pregnancy. This presystolic sound is also heard in patients with first-degree atrioventricular block (prolonged P-R interval). However, an audible fourth heart sound usually indicates heart disease, and its presence is usually dependent on three factors: 1) effective atrial contraction, 2) unimpeded ventricular filling, and 3) diminished ventricular distensibility ("stiff ventricle").

The fourth heart sound is never present in patients with atrial fibrillation and is an uncommon finding in patients with diminished left-ventricular filling due to moderate or severe mitral stenosis. It is usually absent in patients with constrictive pericarditis. The atrial gallop generally signifies reduced ventricular distensibility and is frequently but not always associated with an increase in ventricular end-diastolic pressure.

The presystolic gallop may originate in the right or left ventricle. Left-sided fourth heart sounds are commonly present in patients with diastolic hypertension, severe aortic stenosis, myocardopathies, and acute mitral regurgitation. Most patients with an acute myocardial infarction and sinus rhythm have a prominent fourth heart sound. A presystolic gallop is a frequent finding in patients with coronary artery disease but may be heard only during an episode of angina.

Left-sided fourth heart sounds are frequently accompanied by visible and palpable presystolic distension of the left ventricular apex. This is best observed with the patient on his left side. On phonocardiogram, the low-frequency vibrations of the atrial gallop are coincident with the presystolic "a" wave of the apexcardiogram. The left-sided fourth heart sound is best heard by using light pressure with the bell of the stethoscope and is maximal in intensity at the left ventricular apex with the patient in the left lateral position. If patients are not turned to this position during auscultation, over 50% of atrial gallops will be undetected. The left-sided presystolic gallop is usually most prominent during the expiratory phase of respiration.

The atrial gallop increases in intensity, and the fourth heart sound-first heart sound interval lengthens as the result of an increase in ventricular filling, a prolongation of atrioventricular conduction, or a decrease in ventricular distensibility. During bedside auscultation the left-sided atrial gallop is usually accentuated after coughing and during mild supine exercise. It also becomes prominent during a sustained handgrip contraction. During these maneuvers the fourth heart sound-first heart interval frequently increases in contrast to splitting of the first heart sound which becomes less evident with the increase in heart rate.

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Right-sided fourth heart sounds are frequently present in patients with right-ventricular hypertrophy secondary to either pulmonary hypertension or pulmonary stenosis. They are commonly accompanied by a prominent presystolic "a" wave in the jugular venous pulse and a parasternal or subxiphoid right-ventricular lift. These low frequency sounds are heard best at the third to fifth left intercostal spaces and often increase in intensity during inspiration.

Both the right and left-sided fourth heart sounds can often be distinguished from the two components of the first heart sound by applying increasing chest-wall pressure with the bell piece of the stethoscope. As pressure is increased the bell functions as a diaphragm, and low-frequency sounds such as the fourth heart sound usually decrease in intensity or disappear. In contrast, the high frequency components of the first-heart sound persist unchanged.

Third Heart Sound

The third heart sound (ventricular gallop, protodiastolic gallop) is a low-frequency sound produced in the ventricle in early diastole during passive rapid filling. This early diastolic sound is a frequent finding in normal children and young adults and in patients with a high cardiac output. However, the presence of a third heart sound in patients over the age of 40 generally indicates ventricular decompensation or A-V valve regurgitation. The ventricular gallop, like the fourth heart sound, can be produced in either ventricle and is heard best with the bell piece of the stethoscope. The left-sided third heart sound, commonly present in patients with left-heart failure or mitral regurgitation, is heard best at the left ventricular apex with the patient in the left lateral position. This low-frequency sound is most prominent during expiration. The right-sided ventricular gallop, frequently present in patients with right-heart failure or tricuspid regurgitation, is heard best at the lower left sternal border and increases with inspiration. It is often accompanied by a prominent late systolic "v" wave in the jugular venous pulse, the systolic murmur of tricuspid regurgitation, and a large liver which pulsates in late systole. The third heart sound occurs later in diastole than the higher frequency A-V valve opening snap from which it must be distinguished. The ventricular gallop, unlike the opening snap, decreases or disappears when the patient assumes the upright position.

Risk Disclosure in Experimentation

The failure by a physician fully to disclose to a patient or to the subject of an experiment the risks involved in treatment may well be grounds for a malpractice suit. While most cases have concerned non-disclosure to a patient of risks from treatment which the attending doctor considered necessary, the Department of Health, Education and Welfare (HEW) and the Food and Drug Administration (FDA) recently have devoted considerable attention to the problem of nondisclosure in experiments. In 1971, new regulations were promulgated by HEW governing disclosure to subjects in institutional investigations and by FDA (which has authority to issue regulations on certain matters, including the use of drugs) governing disclosure to subjects given new drugs in any investigation. These regulations, although in terms applying to a limited number of situations, by analogy could affect "experimentation" by doctors neither affiliated with institutional investigation under contract with HEW, nor administering new drugs.

The HEW regulations require that grants or contracts for experimentation involving human subjects be made only after an "institutional committee," which may be a board of directors, medical staff committee, research committee or other standing committee, or a group established solely for project review, has approved the project. The committee "should be able to determine acceptability of the proposal in terms of institutional commitments and regulations, applicable law, standards of professional conduct and practice, and community attitudes," and may be drawn from persons within and without the institution. The committee must carry out initial and continuing review of projects to insure that the rights and welfare of subjects are adequately protected, that the risks to subjects are outweighed by the potential benefits, and that the informed consent of subjects is properly obtained. Risk is defined under the regulations as the possiblity of harm, "physical, physchological, sociological or other."

The FDA also requires institutions to have committees, which must have members of varied backgrounds, to pass on whether the risks to human subjects from the use of investigational new drugs is commensurate with any gain to be derived from such use. The FDA law and regulations governing new drugs further require doctors to obtain an informed consent from any individual to whom investigational drugs are given. There are only two exceptions to this requirement: (1) where, because of inability to communicate, it is not feasible to obtain the patient's or his representative's consent, and it is imperative that the drug be administered without delay; and (2) where it would seriously affect the patient's well-being to communicate the information necessary to obtain consent. It must be noted, however, that both of these exceptions apply only in cases of new drug use for the treatment of disease. Neither of these exceptions applies where "investigational drugs are administered primarily for the accumulation of scientific knowledge."

Under both HEW and FDA regulations, the informed consent of subjects must be obtained. By way of comparison, in cases involving standard treatment for or operations on patients, the disclosure that the courts generally hold must be made to obtain an "informed consent" is that "which is reasonable under the circumstances"—as "reasonable" is interpreted by the court, not necessarily in accordance with prevailing professional practices. In assessing the reasonableness of a disclosure, the court may take into account the seriousness of the impairment risked, the probability of its occurrence, the nature of the illness treated, the character of the treatment, and the relation between doctor and patient. The use of this standard is illustrated by a case recently decided by the United States Court of Appeals for the District of Columbia on May 19, 1972, requiring full disclosure of a one percent risk of paralysis before performance of a laminectomy. The Court held the doctor's failure to reveal a risk of paralysis, although motivated by the doctor's conclusion that disclosing the slight risk might deter the patient from undergoing needed surgery, was a prima facie violation of his duty to disclose.

While in cases involving accepted treatment, such as performance of a laminectomy, courts speak of the reasonableness of disclosure, the HEW and FDA regulations use less flexible terms. By law, the courts must apply the FDA standards to any use of new drugs. If the courts were to apply the same requirements for risk disclosure to experiments generally as HEW now does for experiments by contracting or grantee institutions, then doctors would be required to disclose fully all risks whenever a new form of treatment is used. (An illustration of the use of non-routine treatment to benefit a patient later being attacked as experimentation is the recent suit against Dr. Denton Cooley for allegedly experimenting on a patient by using an artificial heart to keep him alive until a human donor could be found.) It should be noted that deciding not to treat a patient who would conventionally receive some treatment could be considered experimentation.

How courts would apply the full disclosure requirements of the HEW and FDA regulations to experimentation generally is unclear where therapeutic treatments are involved. Under the literal reach of the disclosure requirement, a doctor would have to inform his patient if, failing to ascertain an organic cause for the patient's complaint, he decided to give a placebo instead of a drug normally given for the patient's ostensible ailment. It seems unlikely that the courts would require such self-defeating revelations where the ailment is minor; however, the courts might find that failure to prescribe the drug normally given for such complaints is experimentation, so that the risks—for example, of aggravation of the ailment if an organic cause exists—must be explained to the patient. In resolving this problem, the courts may consider the certainty with which the ailment was or could have been identified, its severity, and the possible benefits from temporary non-disclosure. Since there are medical reasons for prescribing placebos that would be defeated by requiring disclosure, the courts might be hesitant to find disclosure required unless the doctor has identified an organic cause for a complaint of a serious nature and decides that no treatment, instead of the standard treatment, should be given.

Since it is difficult to distinguish ordinary treatments from experimentation where a patient goes to his doctor with a problem and the doctor prescribes a treatment, or decides that none is necessary, as a remedy for the patient's problem, different courts may use different standards (either that standard normally applied to ordinary treatment or the standard applicable to HEW contracting or grantee institutions) to judge disclosures made in similar or even identical circumstances.

Where a doctor engages in experimentation not designed to aid the subject, this problem should disappear. The requirements for disclosure are both more clear and more stringent where the treatment is non-therapeutic. The subject must be informed of the nature of the experiment, its probable duration, the method by which it will be conducted, and all hazards, inconveniences and possible effects upon his health. The subject also must be free to withdraw from participation at any time. It should be observed that at least one case in the District of Columbia has held that a minor, even if mature and informed, cannot legally consent to treatment that is not therapeutic; Maryland and Virginia courts apparently have never ruled on this question. In non-therapeutic experiments, the HEW and FDA regulations

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leave no room for weighing considerations favoring and opposing disclosure of any particular risk in given circumstances. All risks, no matter how slight the possible harm or how unlikely its occurrence, must be revealed to the subject. Possible social inconvenience as well as physical harm must be discussed.

However, even in non-therapeutic experiments, complete disclosure of the risks involved may be made without jeopardizing the results of experiments dependent to some extent on the subjects' ignorance. Where a control group is to be used—for instance, one group given placebos while another is given a new drug—all the participants in the experiment would have to be informed that a certain number of them would be given a harmless substance and others the drug. The control group, however, need not be told that they were given placebos, nor the members of the target group assured that they were given the drug, if such knowledge would affect the experiment.

To ensure that he is protected against claims of failure to comply with the appropriate disclosure requirements [of the FDA, if new drugs are used; of HEW, if the treatment is performed by a grantee under an HEW grant or if courts apply the HEW regulations to experimentation generally; or the customarily-applied requirements, if neither experimentation nor a new drug is involved], each doctor should secure the written consent of the subject or his representative. Such written consent, which in the case of the HEW regulations is required to be approved by the institutional committee and is made mandatory for grantees under most circumstances, should state that the subject has been told and understands the risks involved and should list the risks and their respective probabilities of occurrence.

While the area of liability for nondisclosure of risks remains unsettled, the maximum protection is afforded to the doctor in all circumstances if he makes certain that complete disclosure of all conceivable risks is made and that the subject's consent (or that of the appropriate representative) is obtained before using a non-routine method of treatment, conducting any experiment or administering any new drug.

Edited by Stefan F. Tucker of Arent, Fox, Kintner, Plotkin and Kahn, Washington, D.C.

Medical Expense Deductions. . .

The article entitled "Medical Expense Deductions," which appeared on pages 535 and 536 in the August issue of the Annals (Volume 41, Number 8), the last sentence of the next-to-last paragraph (in describing the rules applicable to the taxation of amounts received by an employee pursuant to a wage continuation plan) inadvertently stated, as a general rule, the rule which applies only to absences commencing prior to January 1, 1964. That sentence should have read as follows: "For absences commencing after December 31, 1963, Danny may exclude from income up to a weekly rate of $75.00 per week for the first 30 calendar days of absence and up to a weekly rate of $100.00 per week after the first 30 calendar days of absence; no exclusion is allowed, however, for the first seven calendar days of absence unless Danny is hospitalized for at least one day during the first seven calendar days of absence."

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Have you ever seen a realistic chart on food cholesterol?

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MEAT, FISH AND EGGS	Cholesterol (mg)
Liver (3½-oz. serving)	300
Eggs (1 large)	275
Oysters (5 to 8)	200
Lobster (3½-oz. serving)	200
Shrimp (10 small)	125
Clams (5 to 10)	99
Veal (3½-oz. serving)	90
Pork (3½-oz. serving)	70
Beef (3½-oz. serving)	70
Lamb (3½-oz. serving)	70
Freshwater fish (3½-oz. serving)	70
Chicken (3½-oz. serving)	60

DAIRY FOODS	Cholesterol (mg)
Whole milk (8-oz. glass)	27
American cheese (1 oz.)	25
Ice cream (¼ pint)	23-34
Heavy cream (1 tbsp.)	17
Creamed cottage cheese (½ cup)	14
Butter (1 pat)	12
Gouda cheese (1 oz.)	10
Yogurt (½ cup)	6
Half and half (1 tbsp.)	5
Skim milk (8-oz. glass)	1

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The Mental Health Committee

Report of the Subcommittee on the Problem of Rape in the District of Columbia

In the District of Columbia, from September 1965 to June 1970, 5,734 complaints of sexual assault were made to the police department. It is well known that the actual number of rapes occurring is much higher than those reported. Our subcommittee has talked with many people who are knowledgeable about this problem and have identified serious gaps in our community services, medical, psychological and legal that are needed by the victims. As members of this community and as professionals concerned with the health needs of our citizens, we have formulated recommendations we wish the full Mental Health Committee to consider on the subject of providing better care for the victims of rape. We will do this by first outlining the multiple steps a woman endures when she is sexually assaulted and state our specific recommendations for each step in the continuum.

The first step is when a woman who is raped notifies the police. Her complaint is relayed to a member of the sex squad who is usually the first helping individual a rape victim encounters. Even if a police cruiser should be the first on the scene, the officer will generally call a member of the sex squad who will respond and escort the victim to the hospital. The function of the sex squad is to investigate complaints of sexual assault and are involved in the case up until the court trial should the case get that far. The public health nurse of the D.C. Sexual Assault Project is assigned to the sex squad but may not make contact with the victim until 24 hours later.

We feel this first step in the need for sympathetic support and reduction of emotional trauma to be the most critical phase for the victim and while we have high regard for the job the sex squad does, we feel their work can be made more effective by the hiring of at least one more nurse or social worker to be assigned to the sex squad and to accompany the members of the sex squad when they respond to the victim and take her to the hospital. Although we do not have evidence to the contrary, we would like to underline very strongly that the police examination, both the timing of it and the exploratory nature of it, take into due account the emotional suffering experienced by the victim.

The second step occurs when the victim goes to the hospital. Although a number of hospitals have recently offered to treat victims of sexual assault, this is not true of all and most victims are still transported to the crowded and overburdened D.C. General Hospital. We recommend and urge that the emergency services of all hospitals, private and public, be made available to treat rape victims. We also recommend that the physical examination be made as humane as possible, including a separate physical facility for the examination and that the examination be done by qualified personnel in the field of obstetrics and gynecology. Their expertise in determining whether a rape has occurred would reduce the sometimes ambiguous medical reports received by the court when the examination is done by interns.

The third step indicates the lack of immediate psychological supportive aid in the emergency room of the hospital. If our earlier recommendation requesting the hiring of another nurse or social worker is not followed or if he or she is not available when the victim goes to the hospital, we recommend that the hospitals then make available a qualified professional to offer emotional support to the sexually assaulted individual. The definition of qualified professional help can be left to the discretion of the hospital administration. In some hospitals, it can be a member of the

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psychiatric staff; in others, a social worker or nurse. It is less important as to the professional status of the person than his or her immediacy to the victim and the supportive help offered. This individual can then be the contact point to the public health nurse and alert her if severe emotional reactions were observed in the victim, thereby making it possible for quicker intervention procedures to be undertaken by the nurse. We also recommend that back up support to the nurse in the form of consultation to her when needed as well as psychiatric treatment of sexually assaulted victims when indicated be provided by both public agencies like community mental health centers as well as by private psychiatrists.

The fourth step concerns the legal system and is one in which our subcommittee recognizes its own lack of expertise in this field as well as the danger of making recommendations for another wholly different agency. But we would be derelict in our duty if we did not press for reform to make the cross examination less demeaning and as humane a procedure as is possible for the rape victim. We advocate and recommend that the U.S. attorney's office take measures that will protect the integrity of the individual during the cross examination. This is especially pertinent for children and adolescent victims. We recognize that in our legal code the accused has a right to face his accuser. But we wonder if justice could not be served equally well while protecting the delicate and unfolding ego processes of young children and adolescents. We take as our model the protection afforded young children in other civilized countries where an adult acting impartially in behalf of the court takes the testimony of the child in private and acts in place of the child during the court proceedings. The child is not present at all in the court. We feel this very civilized procedure makes for good common sense and in no way detracts from justice and truth being served. We would urge the U.S. attorney's office to consider this alternative where youth is involved in sexual assault cases.

Forensic psychiatric experts consider the rapist a potential murderer whose primary purpose is humiliating and physically assaulting a woman, the sexual act being secondary to the wish for violence against women. As recidivism in rapists is high and as most are out on bond until the court hearing, we urge the minimum delay between arraignment and trial.

Lastly, we hope that all organizations with an interest in this serious and rising problem cooperate and coordinate their efforts to prod the slow machinery of progress onward to make the necessary reforms that will benefit all of us living in this community.

Edward H. Weiss, M.D. Chairman
Norman Taub, M.D.
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d.c. HEALTH NOTES

Additional Funding for Mobile Health Screening

Expansion of existing facilities will enable the Community Health and Hospitals Administration to more than triple the number of people who receive health screening tests.

With one mobile screening unit, which moves from neighborhood to neighborhood, and one fixed unit on the grounds of D.C. General Hospital, some 80 persons a day receive screening tests for heart abnormalities, anemia, Sickle Cell Disease, diabetes, renal disease, glaucoma, visual and hearing impairment, venereal disease, and for females, cervical cancer (Pap Smear). The entire testing procedure takes about 45 minutes. Last year, more than 13,000 men and women utilized these facilities.

Table I shows the number of tests completed during 1971, along with the number and percent of abnormal tests. The greatest percentage of abnormal tests are urinalysis (68.7%). It should be noted that the majority of abnormalities in this category are minor and these are not referred to the physician. The next largest percentage of abnormal tests are audiometry (49.6%), and visual acuity (35.6%). The smallest percentage of abnormal tests are chest x-rays for tuberculosis (.2%).

The Social Rehabilitation Services Administration recently approved a request for $450,000 which will finance three additional mobile health trailers. These are scheduled to begin operation this month and will increase the number of persons who can receive health tests to approximately 200 a day. It is estimated that 48,000 people will be screened each year.

The Multiphasic Screening Program is mainly directed toward low income adults age 21 and over. Its purpose is to find health problems early, when treatment is less difficult than in advanced disease. Furthermore, expansion of the program may lessen the need for long term hospital care.

Those persons with abnormal findings are either referred to their family physician for

TABLE I: Number and Percent of Tests Performed by Type of Test, January-December 1971

	Completed		Normal		Abnormal	
Type of Test	Number	%	Number	%	Number	%
Visual Acuity	13,289	100.0	8,564	64.4	4,725	35.6
Audiometry	13,416	100.0	6,759	50.4	6,657	49.6
Blood Pressure	13,400	100.0	11,475	85.6	1,925	14.4
Spirometry	294	100.0	278	94.6	16	5.4
EKG-Six Leads	12,890	100.0	9,783	75.9	3,107	24.1
Tonometry	9,807	100.0	8,323	84.9	1,484	15.1
Head and Neck	6,970	100.0	6,737	96.7	233	3.3
Heart Sounds	6,300	100.0	6,160	97.8	140	2.2
Hemoglobin	12,920	100.0	11,799	91.3	1,121	8.7
Blood Sugar	13,396	100.0	12,456	93.0	940	7.0
B.U.N.	13,374	100.0	10,448	78.1	2,926	21.9
Cholesterol	1,096	100.0	981	89.5	115	10.5
Cervical Smear	2,541	100.0	2,319	91.3	222	8.7
Chest X-Ray						
Tuberculosis	9,819	100.0	9,803	99.8	16	.2
Cardiovascular	9,819	100.0	9,634	98.1	185	1.9
Neoplasm	9,819	100.0	9,739	99.2	80	.8
Other Pulmonary	9,819	100.0	9,074	92.4	745	7.6
Hematocrit	8,232	100.0	7,326	89.0	906	11.0
Serology	13,233	100.0	12,294	92.9	939	7.1
Urinalysis	6,535	100.0	2,045	31.3	4,490	68.7
Cervical Culture	1,501	100.0	1,494	89.5	7	.5

diagnosis and treatment, or to the Community Health and Hospital Administration clinic nearest their residence. Last year, 75 percent of abnormal tests were confirmed on follow-up by physicians.

Three of every five people who took part in the health screening program were referred to physicians for some abnormality. It was estimated that each person referred had approximately two health problems that needed additional study.

The mobile health trailers, specially designed to handle health screening tests efficiently, are staffed by trained health technicians and are under the supervision of a program director and the chief of the Multiphasic Screening Division. Other personnel encourage community participation. We know that persons with a limited income, who have enormous economic and social problems, are less likely to seek preventive health care than more affluent members of the society. Health educators go door to door and meet with men and women in community groups to discuss the need for the tests. Printed material which describes the health tests is distributed. Also, the health testing program is publicized through the news media, radio, and television.

Blood specimens and cultures are sent to the Community Health and Hospitals laboratory for analysis and the results are made available within a short period of time. Public health staff call those with abnormal findings to remind them to make an appointment with their physician, if they have not already done so.

The mobile health trailers are placed in areas with a high medically indigent population. Locations selected are those with high visibility, where large numbers of people congregate. Shopping centers and busy street corners where transportation is good are ideal locations.

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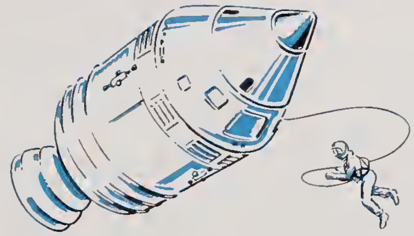

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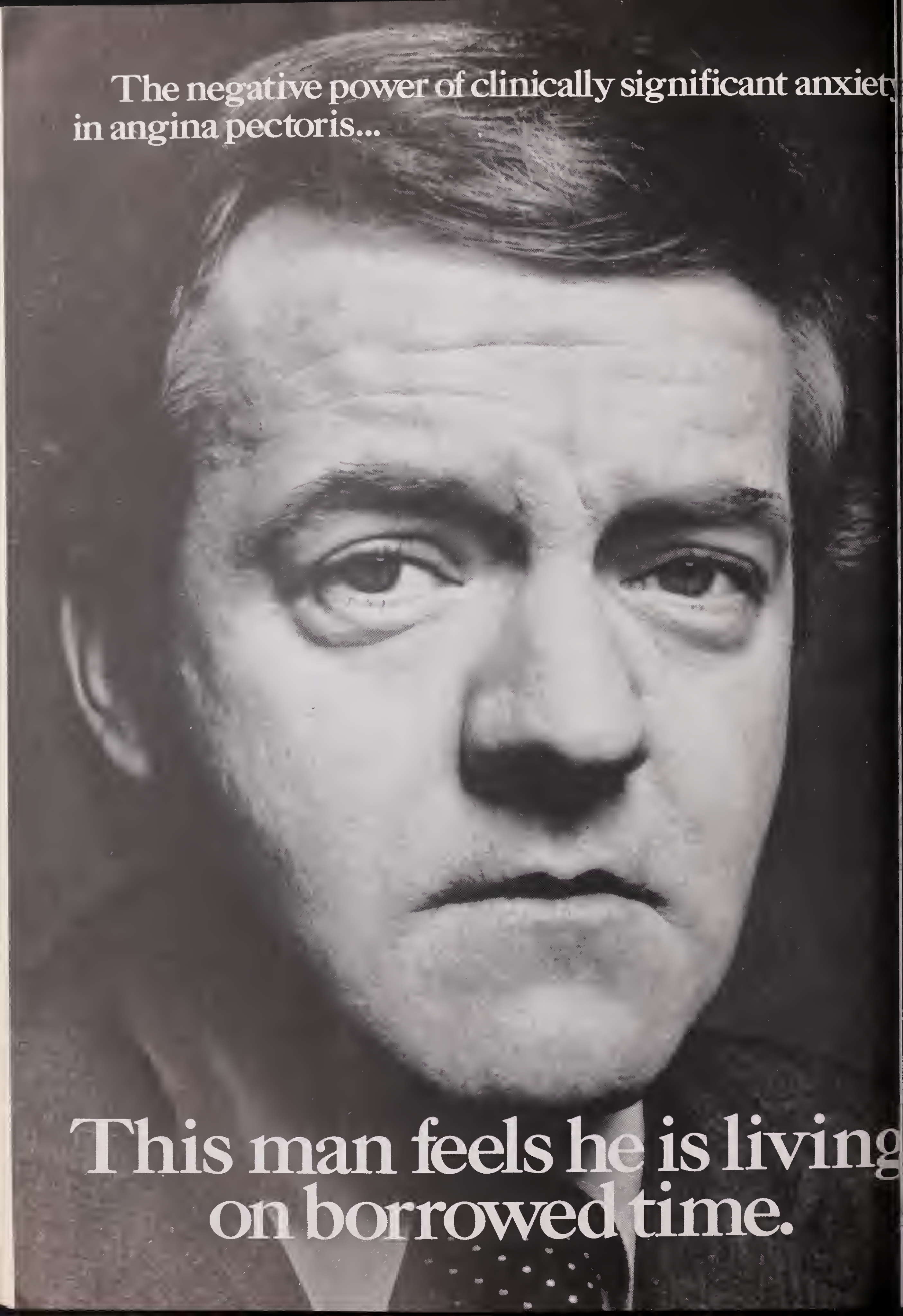
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Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

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*Levine, S.: "Angina Pectoris and Emotional Overlay," Scientific Exhibit presented at the Annual Meeting of the Maine Medical Association, Kennebunkport, Me., June 13-15, 1971.

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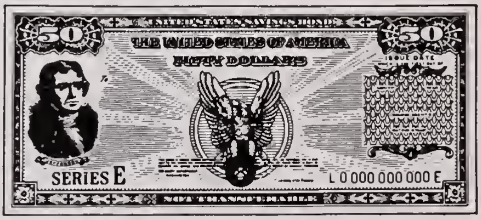

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book REVIEWS

Review of Refraction. JACK HARTSTEIN, M.D. Price, $9.75. Pp. 246 with illustrations. St. Louis: C.V. Mosby Company, 1971.

This book is divided into 24 chapters and takes the question and answer format. The text follows a logical succession from basic optics through retinoscopy and on into more complex lens problems, both theoretical as well as those encountered in everyday clinical practice.

The material is presented in palatable units of information and is subject to easy review at the end of each section. The format allows for a clear understanding of some potentially complicated concepts such as the "Conoid of Sturm," "cyclophoria," and the "accommodative convergence:accommodation" ratio.

This text is designed primarily for the neophyte resident. All training programs would do well to incorporate this book into their teaching sessions. It would also serve as an excellent review by senior residents preparing for the Board Examinations. The veteran Ophthalmologist however, would find little to attract him.

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Drill's Pharmacology in Medicine. JOSEPH R. DI PALMA, M.D., Editor. Price, $25.00. Pp. 1920. New York: McGraw Hill Book Company, 1971.

This is the fourth edition of this well known text. In his preface to the new edition Dr. DiPalma comments on the tendency in medical education today to summarize, to emphasize only the core material in teaching, to move the student to the clinical setting earlier with less in-depth study of various basic disciplines in medicine. Dr. DiPalma states that the new edition has avoided these tendencies and attempts to present as complete a text as possible. A great deal of rewriting as well as adding and subtracting material has had to go into this new edition because of the many advances which have occurred in clinical pharmacology during the past fifteen years, and particularly since the last edition in 1965. New and more logical chapter headings have been formulated, permitting a consolidation from ninety chapters to eighty-eight.

A general review of chapter structures reveals that sub-headings are distinct and well chosen, permitting the reader to arrive at his area of interest without delay. Each chapter has a list of pertinent references attached which are happily selective but not exhaustive. The dates of many of the references are as recent as 1967 and 1969. Chemical formulations of drugs and reproductions of tables and graphs are scattered comfortably throughout each chapter. One is impressed with the conciseness with which the basic pharmacology is presented; clinical application of drugs is dealt with more superficially although in most instances the clinical indications and usages are spelled out clearly. This fact is undoubtedly a reflection of the fact that most of the contributing authors are basically oriented in their own research endeavors. This fact does not detract from the purpose of the text which is to present full coverage of the basic pharmacology of drugs.

In Part I of the text there are five chapters which present the major facets of modern pharmacology including absorption, distribution, and excretion of drugs; a section devoted to the fate of drugs in the body; one section devoted to mechanisms of molecular drug action (discussions of enzyme competitive inhibition and blockade of receptor sites); and finally a section dealing with radioactive tracer techniques. In summary, this segment of the text provides an excellent review of various aspects of pharmacology for the practicing physician.

Two specific examples of what can be derived from this book are presented briefly. First the chapter on Antiarrhythmic Drugs as written by Drs. Brian Hoffman and J. Thomas Bigger, Jr. presents an excellent review of cardiac electrophysiology. This is followed by an excellent coverage of all presently available agents including propranalol and their mechanisms of action. The chapter concludes with a Summary of Clinical Use and a listing of the various agents and their available dosages. Another chapter is entitled Drugs Used in the Treatment of Parkinsonism and is authored by Drs. Roger Duvoisin and Melvin Yahr. One-half of this brief chapter is given to a concise review of Levodopa's application to this disease.

This new edition of Drill's Pharmacology in Medicine is a much matured version of its predecessor editions. The field of pharmacology has grown so fast and has broadened into biochemistry and clinical medicine so much that the preparation of large compendiums with frequent updating and editing have become necessary. This text compares most favorably with the other well known "giant" in the field of Pharmacology, namely Goodman and Gillman's "The Pharmacological Basis for Therapeutics." One is struck by the apparent fact that these two texts, published by different companies, are alternating the preparation and updating of new editions much after the fashion that Harrison and Cecil and Loeb trade honors in Internal Medicine. This is most desirable, and Drill's Pharmacology in Medicine deserves space on the physician's bookshelf.

LOUIS GILLESPIE, JR., M.D.

Current Practice of Biliary Surgery. GABRIEL A. KUNE, M.D. Price, $17.50. Pp. 418 with illustrations. Boston: Little, Brown & Company, 1972.

Dr. Kune, who trained at the Lahey Clinic in Boston, and at Guy's Hospital in London, aims "to present an up-to-date account of the practice of bilary surgery."

Biliary tract disease now is the commonest abdominal condition seen by surgeons, gastroenterologists and radiologists. Operations upon the biliary tract are among the most frequently performed in general hospitals throughout the world.

The volume begins with a precise description of the subtle variations in biliary anatomy. One is then taken in logical and stepwise fashion through the physiologic, radiologic, and pathologic conditions affecting the biliary tract. In this we are aided by many cholangiograms and beautiful illustrations. In addition, there are approximately 1,000 references, largely current.

The author has been careful to be both clinical and practical while presenting older as well as recent approaches to the diagnosis and treatment of biliary tract problems. He carefully reviews the results that should be anticipated with various modes of therapy.

The comprehensiveness of the material is perhaps not so apparent because of the ease with which the book may be read.

The book should be a must for surgeons, and in spite of its title, it will be of great value to students and specialists in radiology and internal medicine.

RICHARD L. FLAX, M.D.

Review of Medical Pharmacology, 3rd edition. FREDERICK H. MEYERS, M.D., ERNEST JAWETZ, M.D. AND ALAN GOLDFIEN, M.D. Price, $8.50. Pp. 688 with illustrations. Los Altos, California: Lange Medical Publications, 1972.

This worthwhile work contains information valuable to student and practitioner. Important data to aid in daily considerations of common problems are readily available. The editors have omitted or abbreviated material of historical or theoretical nature with the object of reducing the size and cost of the book.

The editors state that "the primary objective of this book is to foster a skeptical attitude to all new drug claims and even to suggest to the practicing physician that he should occasionally reexamine his prescribing habits and critically reevaluate drugs he may have been using for years."

A heavy linen paperback cover keeps expenses down. A new edition has appeared at two-year intervals and the editors promise to continue keeping their contribution current in this manner.

M. VAN KINSBERGEN, M.D.

medical annals of the district of columbia

vol. 41 no. 11 november 1972

Diseases of the Gastrointestinal Tract. W. R. O. GOSLINGS, M.D., Editor. Price, $11.75. Pp. 126 with illustrations. Baltimore: Williams & Wilkins Company, 1970.

This book is the published record of a series of lectures in clinical gastroenterology which took place on October 29 and 30, 1969 in Leyden, Netherlands. According to editorial remarks in the preface, the book was designed to be a review for practitioners, presumably acquainting them with recent developments in clinical aspects of gastrointestinal disease. However, the list of active participants included only two internists out of the faculty of 16. The remainder were surgeons, pathologists, radiologists and pharmacologists. The articles themselves when read only by title appeared comprehensive, though not necessarily covering subjects of current interest to clinicians. Those written by the Dutch authors were poorly edited and in translation seemed excessively wordy. Representative titles included "Aspects of the Development and Functional Anatomy of the Stomach", "Primary Carcinoma of the Post-resection Gastric Stump", and "Enzyme Histochemistry of the Small Intestine". The only chapter on esophageal manometry included only two and one-half printed pages and argued against the use of perfusion of pressure measuring catheters and appeared to conclude that the procedure itself was not terribly useful. Their conclusions regarding negative value of continuous catheter perfusion are in almost total variance with the majority of investigators in this field.

Perhaps the book's major defect derives from the superficial way in which the subjects were covered. The chapter on radiology of the biliary tract was confined to the significance of biliary gas and the radiological features of the acute cholecystitis, whereas in the same book, I found a chapter on techniques of measuring gut 5 nucleotidase and DPHN diaphorase before and after gluten application in gluten sensitive enteropathy. It is difficult to conceive of a single audience that would be interested in both of those discussions at the level presented.

In all, the book cannot be recommended for purchase either by physicians or libraries.

DAVID A MOROWITZ, M.D.

The Platelet. K. M. BRINKHOUS, R. W. SHERMER, AND F. K. MOSTOFI, Editors Price, $25.50. Pp. 420 with illustrations. Baltimore: The Williams & Wilkins Company, 1971.

This monograph is an expansion of a lecture series given at the 58th annual meeting of the International Academy of Pathology. Although these lectures were given in 1969, publication of *The Platelet* was delayed almost three years. Unfortunately, this delay has sorely limited the usefulness of this book to the investigator. A monograph should be considered a review of current information, and this work suffers from the same limitations as most standard textbooks in regard to its being up-to-date.

Nevertheless, this book is highly recommended for the student, house officer, practicing internist, and hematologist who desires an overview of the platelet. The chapters on platelet survival, transfusion therapy, and tests of platelet function are particularly useful to the clinician and although somewhat limited by newer information not included–provide a sound foundation for the understanding of platelet kinetics and function.

For those interested in more detailed information regarding ultrastructure, biochemistry, and membrane dynamics, the more up-to-date book entitled *The Circulating Platelet* edited by the late Shirley A. Johnson (Academic Press, New York, 1971) may be consulted. Many of the same contributors have expanded their lectures in Dr. Johnson's book.

The chapters devoted to the platelet disorders reflect our lack of understanding of the role of platelets in many diseases, but a rather complete approach is contained in this monograph with experts in the field presenting well-documented reviews on such diverse subjects as thrombus formation, atherosclerosis, TTP, leukemia, and pseudokalemia. The role of the platelet in DIC is reviewed in depth and most readers will find this chapter enlightening. The discussions of the qualitative platelet disorders suffer from the conflicting data in the literature, but an admirable attempt is made to consolidate the confusing terminology of the "thrombocytopathies."

This monograph is recommended as a supplement to the library of most hematologists, particularly for its clinical application and is suggested as a rather complete reference to the non-hematologist with the aforementioned limitations.

FREDERICK RICKLES, M.D.

Radiography of Infants and Children. 2nd Edition. DONALD B. DARLING, M.D. Price, $28.50. Pp. 211 with illustrations. Springfield, Illinois: The Charles C. Thomas Company, 1971.

The second edition of *Radiography of Infants and Children* by Donald B. Darling, M.D. should prove to be as valuable and as popular as the first.

The backbone of this classic treatise remains the description and instruction in the proper positioning of pediatric patients. Detailed instruction is given for the application of proper restraints, and positioning of the tube. Each procedure is accompanied by photographs of the patient in the properly restrained position along with reproductions of the resultant radiographs.

New sections in neuroradiology and special procedures have been added. In addition, line drawings have been included in the description of techniques for skulls, paranasal sinuses, facial bones, and mastoids. Special studies, not included in the first edition, e.g. dental survey, voiding cystourethrogram, and air contrast barium enemas have been added. Exposure tables are provided throughout the book and summarized in the appendix.

Dr. Darling's book stresses radiation protection and shows the Radiographer how to obtain optimal radiographs with a minimum of radiation exposure—a combination which is of paramount importance in the Pediatric age group. This reviewer congratulates Dr. Darling for his timely contribution. His book should be available in all Radiology Departments that examine infants and children.

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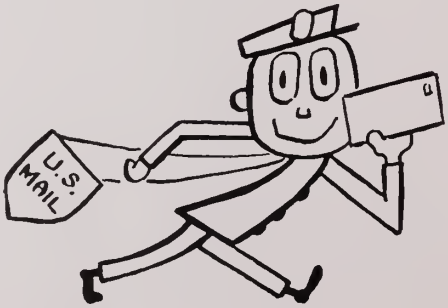

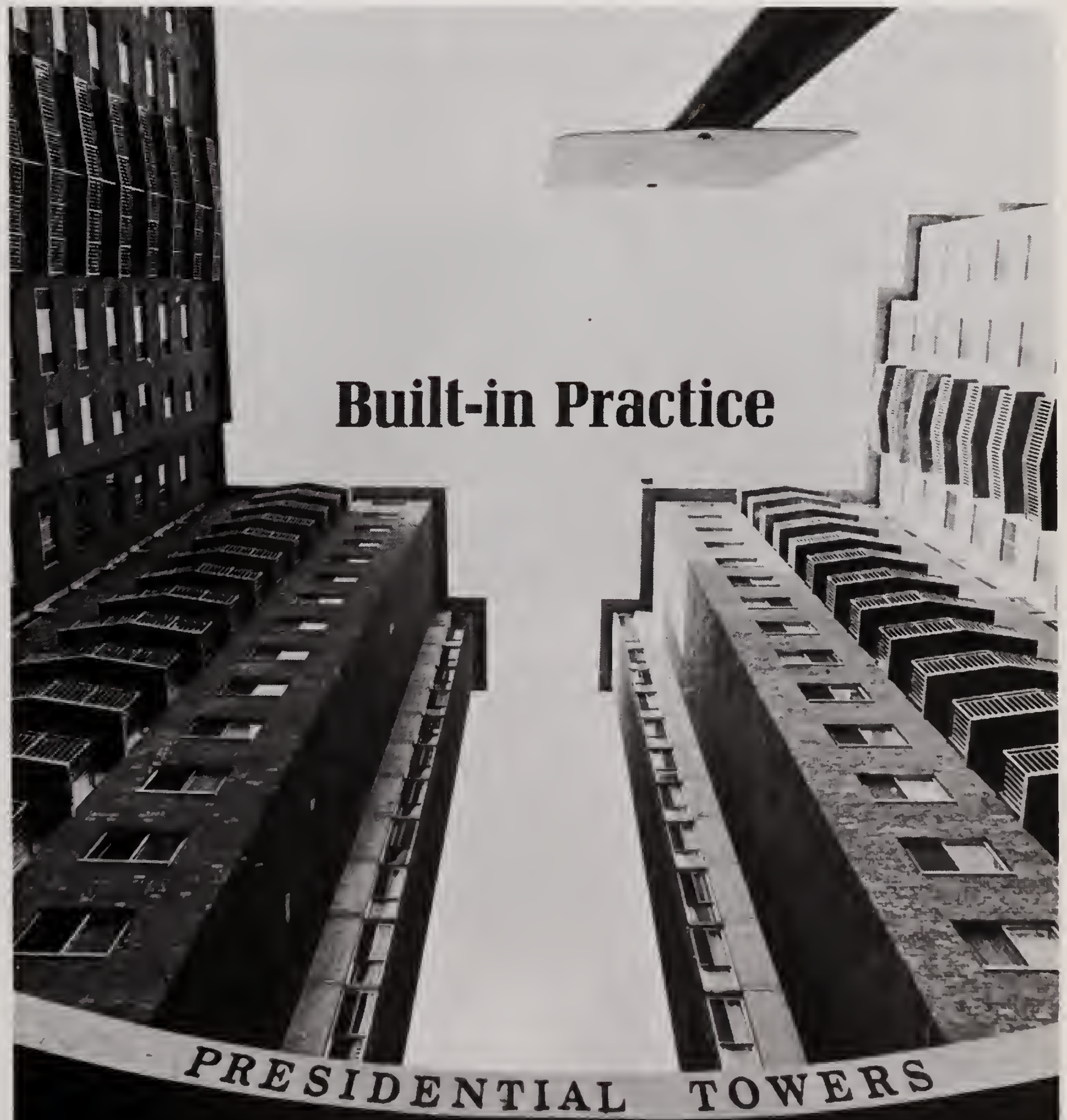

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OBITUARIES

Roger O'Donnell, Jr., M.D.
1904–1972

Dr. Roger O'Donnell, who practiced Obstetrics and Gynecology in the Washington area for 35 years, died August 29, 1972. He resided at 4913-48th Street, N.W.

Born in Buffalo, New York, he was a graduate of the University of Maryland and received his medical degree from the College of Physicians and Surgeons at Columbia University. He served internships at St. Mary's Hospital in Hoboken, New Jersey and at the Sloan Hospital for Women in New York City.

Dr O'Donnell had served on the staff at the old Gallinger Hospital here, and was on the obstetrics and gynecology staff at Washington Hospital Center from its opening in 1958 until his retirement in 1969. Following his retirement he was named staff physician emeritus.

A member of the Medical Society of the District of Columbia since 1935, he was also a member of the American Medical Association.

Dr. O'Donnell is survived by two daughters, Diane M. Morgenthau of Washington, and Ruth O'Donnell of New York City; a brother, Lambert O'Donnell, of Annapolis, and a sister, Adelaide Orme, of Minneapolis.

Etienne Szollosi, M.D.
1905–1972

Dr. Etienne Szollosi, a general practitioner who retired in July of this year from his Maryland practice, died August 10, 1972 in Budapest, Hungary. He resided at 2 Parkway Drive, Forest Heights, Maryland.

Born in Budapest, Hungary, he received his medical degree from the Sorbonne in Paris, where he was awarded the University's bronze medal for an outstanding thesis. Following his internship at the Hospital d'Ambroise in Paris, Dr. Szollosi served with the International Red Cross in China during the Sino-Japanese War. During World War II he served with the U.S. merchant marine.

After the war, Dr. Szollosi came to Washington, D.C. He earned a master's degree in Psychology at George Washington University and became a resident physician at the old Casualty Hospital

He took postgraduate medical studies at New York University School of Medicine and practiced at St. Ann's Hospital in New York City. He started a private practice in Forest Heights, Maryland in 1954, which he maintained until his recent retirement.

An Associate member of the Medical Society of the District of Columbia, Dr. Szollosi was also a member of the Montgomery County Medical Society.

He is survived by his wife, Maria, of the home; a daughter, Helene Dziubanek, of Pasadena, California, and a grandson.

Frank R. Shea, M.D.
1905–1972

Dr Frank R. Shea, a retired Washington area general practitioner, died September 1, 1972. He resided at 4000 Massachusetts Avenue, N.W.

A native of Washington, D.C., he attended St. John's College here and received both his undergraduate and M.D. degrees from Georgetown University. Following his internship at Providence Hospital, he began a practice in General Medicine which he maintained until his retirement in 1970.

A member of the Medical Society of the District of Columbia since 1938, Dr. Shea was also a member of the Georgetown Clinical Society and the Kober Society. He was honored by the Christian Brothers of La Salle College in 1955 and again in 1969 for his services as their physician.

He is survived by his wife, Elmira, of the home; two daughters, Mrs. Casimir Borczuch, of Bowie, Maryland, and Mrs. Philip Truex, of Simi Valley, California; three sisters, Arleen Zahn and Rita Shea, both of Washington, and Mrs. Gibbons Burke, of New Orleans; two brothers, Dennis C. Shea, of Westfield, New Jersey, and Brandan B. Shea, of Falls Church, Virginia, and four grandchildren.

Leland E. Stevenson, M.D.
1900–1972

Dr Leland E. Stevenson, who practiced general medicine in the Washington area for 40 years, died September 14, 1972. He resided at 5101 39th Street, N.W.

Born in Salt Lake City, Utah, he received his undergraduate education at the University of Utah, and received his M.D. degree from George Washington University here. He served his internship and residency at the old Emergency Hospital, and had been on the staffs of Emergency, Doctors and Sibley Hospitals. Dr. Stevenson was also engaged in private practice at 2101 R Street, N.W.

A Life member of the Medical Society of the District of Columbia, Dr. Stevenson also held membership in the George Washington University Medical Society, of which he was a past president. He was also a member of the American Medical Association.

He is survived by his wife, Fay Louise, of the home, and a son, Leland, Jr.

Casimir Leibell, M.D.
1888–1972

Dr. Casimir Leibell, a retired Washington area radiologist, died August 8, 1972. He resided at 2800 Woodley Road, N.W.

Born in New York, New York, he received his M.D. degree from Georgetown University here, and served his internship at Providence Hospital.

Having served in the Army Medical Corps during World War I, Dr. Leibell began his practice in Radiology here in 1919. He had been a Radiotherapist at Providence Hospital and at old Gallinger Municipal Hospital. He had also been a Clinical Assistant Professor of Radiology at Georgetown University School of Medicine and was made Emeritus Clinicial Professor of Radiology upon his retirement in 1956.

Dr. Leibell was a Life member of the Medical Society of the District of Columbia.

He is survived by his wife, Theresa, of the home address.

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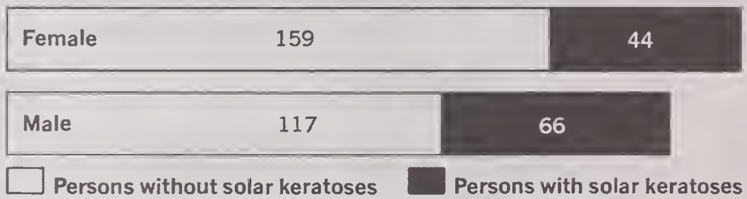
What it means to live and work in Tipton County, Tennessee

Persons who are white and over 40 have one chance in four of having solar keratoses... which may be premalignant

An epidemiologic study* conducted in Tipton County, Tennessee, revealed that 28.5% of white persons over 40 had solar keratoses; most had multiple lesions. Cluster sampling projected an estimated prevalence of 32.5% for white males and 19.5% for white females.

Though this is an unusually high percentage of affected persons, these lesions can occur in any white population, wherever people work or play out of doors.

Prevalence of solar keratoses in white persons over 40 in Tipton County, Tennessee



*Data on file, Hoffmann-La Roche Inc., Nutley, New Jersey.

olar, actinic, senile keratoses

alled by many names, the typical lesion is flat
slightly elevated, brownish or reddish in
lor, papular, dry, adherent, rough, sharply
fined; usually multiple lesions, chiefly on
posed portions of the skin.

equence/selectivity of response

rythema in areas of lesions may begin after
veral days of therapy; height of reaction
nly in affected areas)* usually occurs within
o weeks, declining after discontinuation of
erapy. Since this response is so predictable,
sions that do not respond should be biopsied
rule out the presence of a frank neoplasm.

osmetic results

osmetic results are highly favorable. Inci-
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Precautions: If applied with fingers, wash hands immediately. Apply with care near eyes, nose and mouth. Lesions failing to respond or recurring should be biopsied.
Adverse Reactions: Local—pain, pruritus, hyperpigmentation and burning at application site most frequent; also dermatitis, scarring, soreness and tenderness. Also reported—insomnia, stomatitis, suppuration, scaling, swelling, irritability, medicinal taste, photosensitivity, lacrimation, leukocytosis, thrombocytopenia, toxic granulation and eosinophilia.
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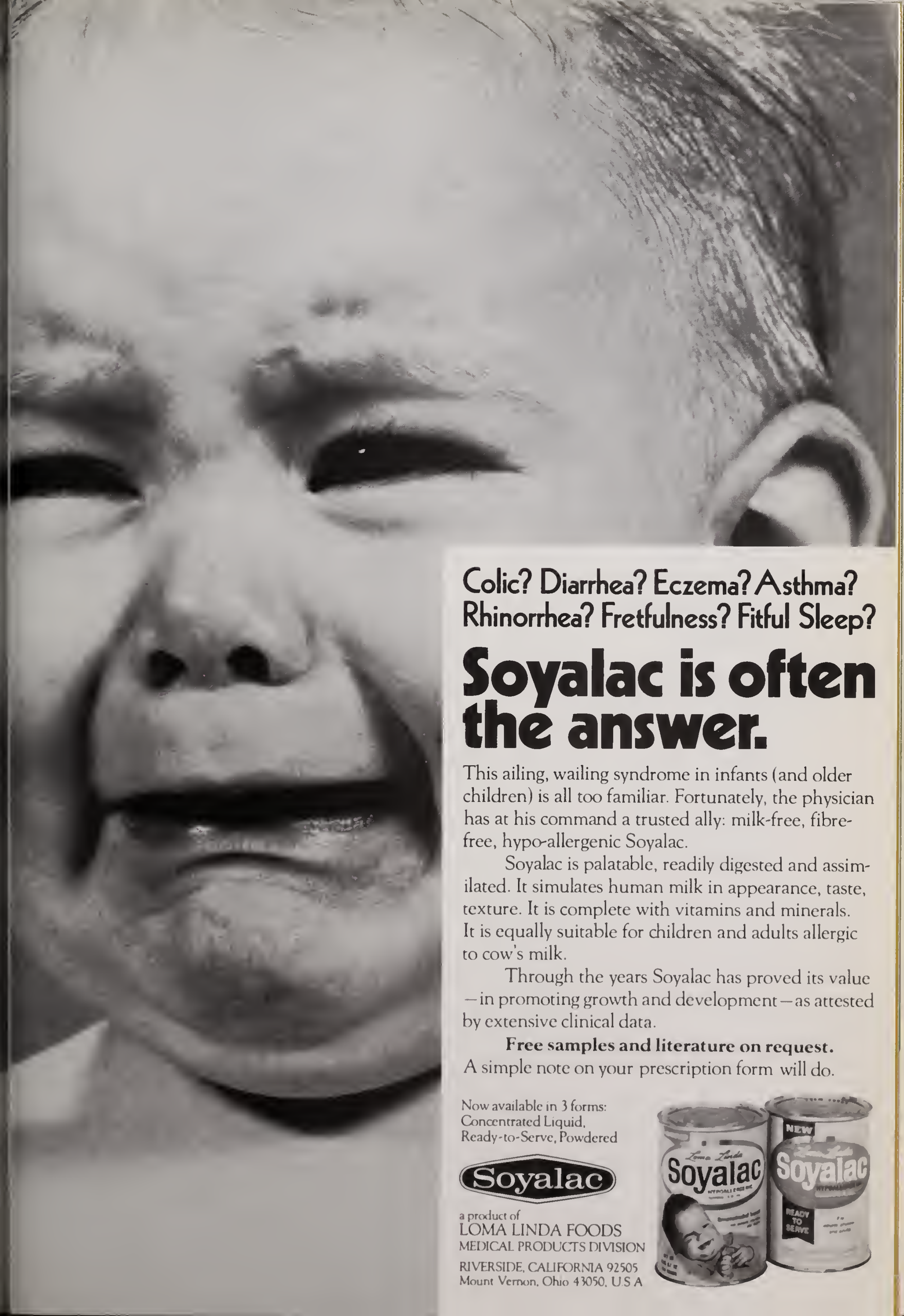

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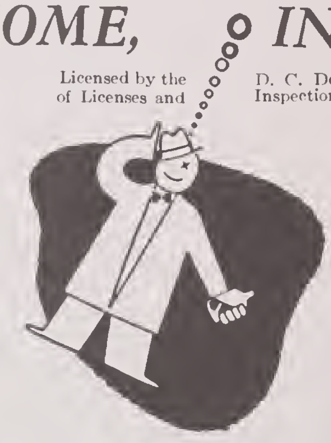

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MEDICAL ANNALS

of the district of columbia

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~~DISPLAY SHELVES~~

WALLACE M. YATER, M.D.
Editor, 1932-1972
Editor Emeritus, January 1, 1973
(See page 3 of the Editorial section)

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MEDICAL ANNALS
of the district of columbia

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THE FRANCIS A. COUNTWAY
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PRESIDENT'S MESSAGE

Well done, good and faithful servant;
. . . enter into the joy of thy Lord.

Darrell C. Crain, M.D.

Such is the approbation recorded by the Apostle Matthew reserved for those who have used well their God-given talents for alleviating the sufferings of mankind.

And surely, in the Valhalla of Asclepian heroes there will be a place for those physicians of our Society, who during the past year have given so much of their time and devotion to the work of our Society in improving the health care of our citizens:

Donald Glew and the Drug Abuse Committee, whose monumental work on methadone and amphetamines has set a pattern for physicians' actions through the country;

Lawrence Summerfield and the Aviation Committee with Harry Zehner and the Emergency Medicine Committee, who have formulated a plan for helicopter-ambulance service, also a model for other communities;

William Kurstin and his devoted chairmen, who made the Greenbrier Assembly an outstanding success;

Paul Chodoff and the Mental Health Committee, whose stand on gun control brought our Society national recognition at the AMA Convention;

James Curtin and his special Venereal Disease Control Committee who worked with Raymond Standard, John Pate and others of our city to bring the scourge of venereal disease to the attention of our citizens;

Howard Silby and the Alcohol Control Committee, who have helped doctors and others in our community with this problem;

J. Lawn Thompson and the Committee on Provision for Health Care who blazed a new trail in the study of this complex problem;

Raymond Scalettar and his editorial staff who brought new life to the ANNALS;

James Kehoe and his staff, who kept our Society abreast of the news.

Edwin Parker and the Utilization and Peer Review Committee, who fairly adjudicated so many questions about medical practice;

Lou Brott, our Public Relations officer, who presented our society so favorably to the public;

Roland Scott and the Child Welfare Committee, who educated us on the matter of sickle cell anemia;

Symposium on Strabismus (Burian et al) reviewed by Thomas Frey, M.D.

Histopathologic Spectrum of Regional Enteritis and Ulcerative Colitis (Mottet) reviewed by J. Chandler Smith, M.D.

Living with a Man Who is Dying (Evans) reviewed by Norman K. Bohrer, M.D

Emergency Medical Manual (Moore) reviewed by Martin I. Cohen, M.D.

Practical Electrocardiography (Marriott) reviewed by Lawrence Klein, M.D.

Identification of Enterobacteriaceae (Edwards and Ewing) reviewed by Lcdr. Robert P. Feinstein, MC, USNR

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For Subscription and Advertising information, see Contents.

William McCune and the members of the Executive Committee, who guided us so skillfully through this turbulent year;

And last but not least Franc Ferraraccio and his staff, who so efficiently kept the wheels of the Society moving;

And more than two dozen other chairmen and hundreds of committee members who have filled our conference rooms daily, discussing every problem from light bulbs in our building to the dangers of lead paint on ghetto dwelling walls.

But special mention must be made of one of our members, a former President, Chairman of the Board, chairman of many committees and Editor of the MEDICAL ANNALS who is now retiring, Wallace M. Yater. A tribute to this distinguished colleague appears following this message, but the President would be remiss if he did not express personal as well as Society tribute to a man whose career has been recognized locally, nationally and internationally. Medicine is richer because of Wallace Yater's contributions. We are fortunate that he worked among us.

Well done, good and faithful physicians, all.

Darrell C. Crain

Dr. Wallace M. Yater, Editor Emeritus

On January 1, 1973 Dr. Wallace M. Yater will be elevated to the position of Editor Emeritus of the MEDICAL ANNALS OF THE DISTRICT OF COLUMBIA. Too often such events go unnoticed or unremembered. Dr. Yater's contributions to our Society, to the scientific literature, and to medical practice in general have made him one of the greatest physicians in Washington's long medical history. We shall continue to call upon him frequently for advice and leadership.

Born in Canton, New York, he received a Bachelor of Arts degree cum laude from George Washington University and a Doctor of Medicine degree from Georgetown University School of Medicine. His training years were spent at Garfield Memorial and Georgetown University Hospitals. In 1922 he entered practice. He held many teaching positions leading, in 1930, to the post of Professor and Director of the Department of Medicine at Georgetown, which he held until 1945. At present he is in active practice as Chairman of the Department of Medicine and General Chairman of the Yater Clinic in Washington.

Dr. Yater is best known among his students as a bedside clinician and diagnostician. He taught the importance of logic in diagnosis, and stressed the importance of a student drawing the proper deductions from the facts which he had accumulated.

His contributions to medicine are reflected in a rich bibliography which includes among other subjects studies in cardiovascular disease, hyperthyroidism, renal function, cirrhosis, and malignancy. One of the first to perform angiography for diagnosis, he also succeeded in experimental transplantation of the heart as early as 1932.

Our greatest debt to Dr. Yater, however, is in his founding of the MEDICAL ANNALS OF THE DISTRICT OF COLUMBIA in 1932, and his great devotion to our journal for over forty years. He resolved that the ANNALS would remain a professional journal as well as an official organ of our Society, unlike its predecessor the "Bulletin" which only recorded Society activities from 1924 to 1932. Through many hours of patient, painstaking work for almost half a century he has kept this resolve. Today the MEDICAL ANNALS continues to be a well recognized, respected medical journal of which we can be justly proud.

Many thanks and best wishes to Dr. Wallace M. Yater!

William S. McCune, M.D.

The Physician and the Public

Once again, it appears too late to shut the barn door.

The public relations situation now faced by physicians is one that has been anticipated for decades by organized medicine as well as by each one of us. The recent local newspaper publicity brings such into clear focus for us.

Why?

We, as physicians, have allowed all aspects of medical care to come under lay control.

Hospitals have become burgeoning personnel pools where public relations, complicated and duplicated record maintenance and multitudenous committee meetings appear to have taken equal status with patient care.

Politicians, always looking for re-election propaganda, have grabbed the impetus from the physicians in demanding comprehensive and adequate medical care for the public.

Lay organizations - insurance companies, welfare officers and organizations, unions - in attempting self perpetuation through justification, have gone to the public medium ahead of us.

Thus, the cost of medical care has soared, the quest of the patient for adequate medical care has become mired in the complicated approach, i.e., hospital pre-admission routines and down payments and insurance clearance; closed panel preregistration, rules and regulations; private sector delays due as much or more to the time and energy demands of endless record keeping, reports and meetings on the private physician and his personnel. Too, the eternal quest of each physician for better patient care and more information and education has become inundated in the bureaucracy of hospitals and insurance companies along with the soaring costs of personnel, equipments, rents and malpractice insurance - all this coupled with the fact of only 24 hours in a day.

Can't we, individually and collectively, attempt more active participation in the public relations arena? Who can better represent the consumer, i.e., the patient, against insurance companies and hospitals? Who is better informed about medical and environmental needs and the impediments to adequate medical care than the physician? Who better knows the alleged waste and inefficiency of hospitals and medical insurance companies than the physician?

We must utilize our Medical Society's committees and projects: health planning, hospital and insurance company liaison, continuing education, fee review, grievance, yes and public relations, as well as demand that our officers and committees actively and accurately represent us and keep us informed as to recommended and pertinent policies and procedures dealing with patients, hospitals, insurance carriers, and yes, newspapers.

We must insist on the implementation of the Joint Commission recommendation of more physician participation on policy-making hospital boards.

We must demand that those insurance carriers advertising physician participation be truly responsible to patient and physician through the mechanisms of external audits, implementation of necessary and adequately covered services, and elimination of the "fine print."

The time is now! And the question is, who can best represent the patient?

M.L.B.

They laughed when I bought my first Bond.

In my neighborhood everyone was poor. Most of us were lucky if we had enough food to eat or clothes to wear. So like most kids we spent a lot of time talking about the things we wanted out of life. Things like owning a big car, wearing fine clothes and going to all of the best places. Boy, we could hardly wait to grow up.

Like the other kids I had little odd jobs in grammar school but I got my first real job when I started high school. I was a junior clerk in a big department store. That's where I first heard about U.S. Savings Bonds. My boss asked me if I wanted to join the Payroll Savings Plan and I said yes. A month later I got my first bond.

When my friends found out they really had a good laugh. Imagine buying U.S. Savings Bonds when there were so many other great things to buy. Well, they kept laughing and I kept saving all through high school.

After graduation some of us wanted to go to college. And that's when my Bonds really came in handy. They not only helped pay my tuition, but also helped buy some books.

Well, I'm a senior now and looking forward to graduation. Unfortunately, a lot of my friends never even got started. And to think, they laughed when I bought my first Bond.

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Now E Bonds pay 5½% interest when held to maturity of 5 years, 10 months (4% the first year). Bonds are replaced if lost, stolen, or destroyed. When needed they can be cashed at your bank. Interest is not subject to state or local income taxes, and federal tax may be deferred until redemption.

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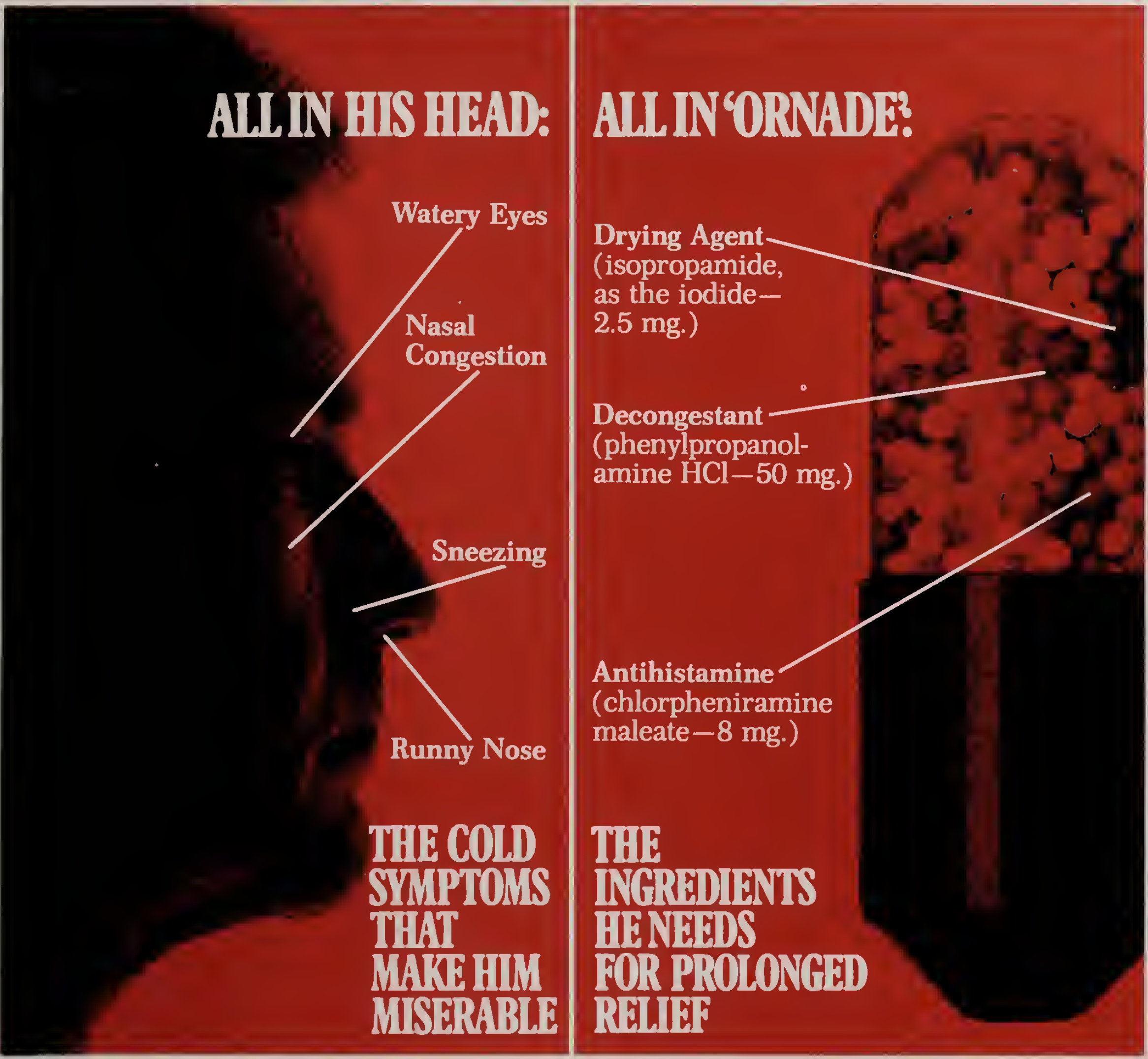
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Contraindications: Hypersensitivity to any component; concurrent MAO inhibitor therapy; severe hypertension; bronchial asthma; coronary artery disease; stenosing peptic ulcer; pyloroduodenal or bladder neck obstruction. Children under 6.

Warnings: Advise vehicle or machine operators of possible drowsiness. Warn patients of possible additive effects with alcohol and other CNS depressants.

Usage in Pregnancy: In pregnancy, nursing mothers and women who might bear children, weigh potential benefits against hazards. Inhibition of lactation may occur.

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Adverse Reactions: Drowsiness, excessive dryness of nose, throat or mouth; nervousness; or insomnia. Also, nausea, vomiting, epigastric distress, diarrhea, rash, dizziness, weakness, chest tightness, angina pain, abdominal pain, irritability, palpitation, headache, incoordination, tremor, dysuria, difficulty in urination, thrombocytopenia, leukopenia, convulsions, hypertension, hypotension, anorexia, constipation, visual disturbances, iodine toxicity (acne, parotitis).

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UNCOMMON RELIEF FOR COLD SYMPTOMS

If you've seen one, have you really seen them all?

The following patient profiles represent typical clinical situations, but do not necessarily represent actual cases.

Age 22, previously normal menses with occasional menorrhagia. Now on a sequential O.C. for four mont
Complains of heavy flow, occasional intracyclic bleeding, edema, tender swollen breasts.

Indicates estrogen excess.

1st choice: Switch to a combination 50-mcg.-estrogen O.C. (such as **Demulen***).

Age 19, small breasts, minor hirsutism, oily hair and skin. History of metrorrhagia, skipped or scanty menses. New user.

Indicates androgenic excess or estrogen deficiency (fertility is suspect).

1st choice: An estrogen-dominant O.C. (such as **Enovid-E***).

Age 25, average frame, poor complexion. No problem with menses, normal para 1. On a low-estrogen/high-progestogen O.C. for two years. Now complains of scanty flow, decreased libido, depression.

Indicates probable buildup of progestogen-related side effects.

1st choice: Switch to a center-spectrum O.C. with more estrogen, less progestational activity (such as **Ovulen***).

Age 21, short, mammose, with normal menses, some acne. Was put on prenuptial regimen of 50-mcg.-estrogen/moderate-progestogen O.C. for two months. Now has increased acne.

Indicates metabolic production of androgen or relative estrogen deficiency.

1st choice: Switch to a 100-mcg.-estrogen combination (such as **Enovid-E*** or a sequential).

Unmasked, physiologically and anatomically, they're not all the same. A basic difference lies in their hormone profiles. One may secrete too much estrogen, another not enough...or perhaps too much androgen; the vast majority would fit somewhere into the broad center spectrum.

Although the profiles described below may not be completely predictive, in optimal O.C. selection, the estrogen-progestogen activity ratio should be carefully matched to the patient profile. Searle offers you O.C.s in a range not only suitable for your patients in the balanced center spectrum, but also adaptable to the patient with another type of hormone profile.

Oral contraceptives are complex medications. Among the commonly reported adverse reactions are: intracycle bleeding, fluid retention, tender or swollen breasts, exacerbation of acne condition, changes in libido, amenorrhea while on medication and upon discontinuance, nausea, leg cramps, headaches, weight gain. Therefore, after reference to the prescribing information, oral contraceptives should be prescribed with care.

*Note: In some patients any level of exogenous estrogen or progestogen may produce symptoms of excess hormone activity.

ge 25, tall, slender, athletic, th flat chest. On a progestogen-ominant 50-mcg.-estrogen O.C. as recurrent trichomoniasis id Monilia.
Indicates estrogen deficiency and cess of progestogen in current O.C.
1st choice: Switch to a com-nation pill with 100 mcg. trogen and less progestational tivity (such as **Enovid-E**® or **ulen**® or a sequential).

Age 23, "Miss America" figure, previously normal menses, healthy skin and hair. On a 50-mcg.-estrogen pill for four months. Complains of intracyclic bleeding.
Indicates probable need for more estrogen.
1st choice: Switch to a center-spectrum O.C. with more estrogen and moderate progestogen dominance (such as **Ovulen**®).

Age 21, college senior, average build. On highly progestogen-dominant/low-dose-estrogen O.C. for six months. Now complains of amenorrhea, between-cycle headaches, weight gain.
Indicates probable progestogen excess.
1st choice: Switch to a center-spectrum pill (such as **Ovulen**®).

Age 27, slightly overweight, multiparous. Nausea with all three pregnancies and with a sequential O.C. three years ago. Has premenstrual fluid retention and leg cramps.
Indicates probable excess of estrogen.
1st choice: A 50-mcg.-estrogen/progestogen-dominant pill (such as **Demulen**®).

Ovulen® a balanced center-spectrum O.C. for most
Each white tablet contains ethynodiol diacetate 1 mg./mestranol 0.1 mg.

Demulen® a moderately progestogen-dominant O.C. for many
Each white tablet contains ethynodiol diacetate 1 mg./ethinyl estradiol 50 mcg.
Each pink tablet in Ovulen-28® and Demulen®-28 is a placebo, containing no active ingredients.
Both Ovulen and Demulen are available in 21- and 28-pill schedules.

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Enovid-E® a moderately estrogen-dominant O.C. for some
Each tablet contains norethynodrel 2.5 mg./mestranol 0.1 mg.

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Where "The Pill" Began

a brief summary
rescribing information,
ase see next page.

a family of O.C. products to help you match the right pill to the right patient

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Each white tablet contains
ethynodiol diacetate 1 mg./mestranol 0.1 mg.

Demulen®

Each white tablet contains
ethynodiol diacetate 1 mg./ethinyl estradiol 50 mcg.

Each pink tablet in Ovulen-28® and Demulen®-28 is a placebo, containing no active ingredients.

Actions – Ovulen and Demulen act to prevent ovulation by inhibiting the output of gonadotropins from the pituitary gland. Ovulen and Demulen depress the output of both the follicle-stimulating hormone (FSH) and the luteinizing hormone (LH).

Special note – Oral contraceptives have been marketed in the United States since 1960. Reported pregnancy rates vary from product to product. The effectiveness of the sequential products appears to be somewhat lower than that of the combination products. Both types provide almost completely effective contraception.

An increased risk of thromboembolic disease associated with the use of hormonal contraceptives has now been shown in studies conducted in both Great Britain and the United States. Other risks, such as those of elevated blood pressure, liver disease and reduced tolerance to carbohydrates, have not been quantitated with precision.

Long-term administration of both natural and synthetic estrogens in subprimate animal species in multiples of the human dose increases the frequency of some animal carcinomas. These data cannot be transposed directly to man. The possible carcinogenicity due to the estrogens can be neither affirmed nor refuted at this time. Close clinical surveillance of all women taking oral contraceptives must be continued.

Indication – Ovulen and Demulen are indicated for oral contraception.

Contraindications – Patients with thrombophlebitis, thromboembolic disorders, cerebral apoplexy or a past history of these conditions, markedly impaired liver function, known or suspected carcinoma of the breast, known or suspected estrogen-dependent neoplasia and undiagnosed abnormal genital bleeding.

Warnings – The physician should be alert to the earliest manifestations of thrombotic disorders (thrombophlebitis, cerebrovascular disorders, pulmonary embolism and retinal thrombosis). Should any of these occur or be suspected the drug should be discontinued immediately.

Retrospective studies of morbidity and mortality conducted in Great Britain and studies of morbidity in the United States have shown a statistically significant association between thrombophlebitis, pulmonary embolism, and cerebral thrombosis and embolism and the use of oral contraceptives. There have been three principal studies in Britain[1-3] leading to this conclusion, and one[4] in this country. The estimate of the relative risk of thromboembolism in the study by Vessey and Doll[3] was about sevenfold, while Sartwell and associates[4] in the United States found a relative risk of 4.4, meaning that the users are several times as likely to undergo thromboembolic disease without evident cause as nonusers. The American study also indicated that the risk did not persist after discontinuation of administration and that it was not enhanced by long-continued administration. The American study was not designed to evaluate a difference between products. However, the study suggested that there might be an increased risk of thromboembolic disease in users of sequential products. This risk cannot be quantitated, and further studies to confirm this finding are desirable.

Discontinue medication pending examination if there is sudden partial or complete loss of vision, or if there is a sudden onset of proptosis, diplopia or migraine. If examination reveals papilledema or retinal vascular lesions medication should be withdrawn.

Since the safety of Ovulen and Demulen in pregnancy has not been demonstrated, it is recommended that for any patient who has missed two consecutive periods pregnancy should be ruled out before continuing the contraceptive regimen. If the patient has not adhered to the prescribed schedule the possibility of pregnancy should be considered at the time of the first missed period.

A small fraction of the hormonal agents in oral contraceptives has been identified in the milk of mothers receiving these drugs. The long-range effect to the nursing infant cannot be determined at this time.

Precautions – The pretreatment and periodic physical examinations should include special reference to the breasts and pelvic organs, including a Papanicolaou smear since estrogens have been known to produce tumors, some of them malignant, in five species of subprimate animals. Endocrine and possibly liver function tests may be affected by treatment with Ovulen or Demulen. Therefore, if such tests are abnormal in a patient taking Ovulen or Demulen, it is recommended that they be repeated after the drug has been withdrawn for two months. Under the influence of progestogen-estrogen preparations preexisting uterine fibromyomas may increase in size. Because these agents may cause some degree of fluid retention, conditions which might be influenced by this factor, such as epilepsy, migraine, asthma, cardiac or renal dysfunction, require careful observation. In breakthrough bleeding, and in all cases of irregular bleeding per vaginam, nonfunctional causes should be borne in mind. In undiagnosed bleeding per vaginam adequate diagnostic measures are indicated. Patients with a history of psychic depression should be carefully observed and the drug discontinued if the depression recurs to a serious degree. Any possible influence of prolonged Ovulen or Demulen therapy on pituitary, ovarian, adrenal, hepatic or uterine function awaits further study. A decrease in glucose tolerance has been observed in a significant percentage of patients on oral contraceptives. The mechanism of this decrease is obscure. For this reason, diabetic patients should be carefully observed while receiving Ovulen or Demulen therapy. The age of the patient constitutes no absolute limiting factor, although treatment with Ovulen or Demulen may mask the onset of the climacteric. The pathologist should be advised of Ovulen or Demulen therapy when relevant specimens are submitted. Susceptible women may experience an increase in blood pressure following administration of contraceptive steroids.

Adverse reactions observed in patients receiving oral contraceptives – A statistically significant association has been demonstrated between use of oral contraceptives and the following serious adverse reactions: thrombophlebitis, pulmonary embolism and cerebral thrombosis.

Although available evidence is suggestive of an association, such a relationship has been neither confirmed nor refuted for the following serious adverse reactions: neuro-ocular lesions, e.g., retinal thrombosis and optic neuritis.

The following adverse reactions are known to occur in patients receiving oral contraceptives: nausea, vomiting, gastrointestinal symptoms (such as abdominal cramps and bloating), breakthrough bleeding, spotting, change in menstrual flow, amenorrhea during and after treatment, edema, chloasma or melasma, breast changes (tenderness, enlargement and secretion), change in weight (increase or decrease), changes in cervical erosion and cervical secretions, suppression of lactation when given immediately post partum, cholestatic jaundice, migraine, rash (allergic), rise in blood pressure in susceptible individuals and mental depression.

Although the following adverse reactions have been reported in users of oral contraceptives, an association has been neither confirmed nor refuted: anovulation post treatment, premenstrual-like syndrome, changes in libido, changes in appetite, cystitis-like syndrome, headache, nervousness, dizziness, fatigue, backache, hirsutism, loss of scalp hair, erythema multiforme, erythema nodosum, hemorrhagic eruption and itching.

The following laboratory results may be altered by the use of oral contraceptives: hepatic function: increased sulfobromophthalein retention and other tests; coagulation tests: increase in prothrombin, Factors VII, VIII, IX and X; thyroid function: increase in PBI and butanol extractable protein bound iodine, and decrease in T^3 uptake values; metyrapone test and pregnanediol determination.

References: 1. Royal College of General Practitioners: Oral Contraception and Thrombo-Embolic Disease, J. Coll. Gen. Pract. *13*:267-279 (May) 1967. **2.** Inman, W. H. W., and Vessey, M. P.: Investigation of Deaths from Pulmonary, Coronary, and Cerebral Thrombosis and Embolism in Women of Child-Bearing Age, Brit. Med. J. *2*:193-199 (April 27) 1968. **3.** Vessey, M. P., and Doll, R.: Investigation of Relation Between Use of Oral Contraceptives and Thromboembolic Disease. A Further Report, Brit. Med. J. *2*:651-657 (June 14) 1969. **4.** Sartwell, P. E.; Masi, A. T.; Arthes, F. G., Greene, G. R., and Smith, H. E.: Thromboembolism and Oral Contraceptives. An Epidemiologic Case-Control Study, Amer. J. Epidem. *90*:365-380 (Nov.) 1969.

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Actions – Enovid-E acts to prevent ovulation by inhibiting the output of gonadotropins from the pituitary gland. Enovid-E depresses the output of both the follicle-stimulating hormone (FSH) and the luteinizing hormone (LH).

Indication – Enovid-E is indicated for oral contraception.

The *Special Note, Contraindications, Warnings, Precautions* and *Adverse Reactions* listed above for Ovulen and Demulen are applicable to Enovid-E and should be observed when prescribing Enovid-E.

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brand of norethynodrel with mestranol

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Where "The Pill" Began

> *"The history of science, and in particular the history of medicine . . . is . . . the history of man's reactions to the truth, the history of the gradual revelation of truth, the history of the gradual liberation of our minds from darkness and prejudice."*
>
> *– George Sarton, from "The History of Medicine Versus the History of Art"*

Are combination drug products useful in treatment involving concomitant use of two or more drugs?

Opinion

Results of a questionnaire to 7,000 physicians:

62.9%
Believe combination drug products are useful.

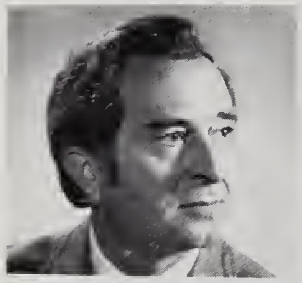
13.8%
Do not believe combination drug products are useful.

Are combination drug product useful in treatment involvin concomitant use of two or more drugs

Opinion & Dialogue
Dialogu

Doctor of Medicine

Louis Lasagna, M.D.
Professor and Chairman
Department of
Pharmacology & Toxicology
University of Rochester
School of Medicine
and Dentistry



Obviously, many drugs are given concomitantly. Whether it makes sense to combine medications in one preparation, be it capsule, tablet, or liquid, is a question that can be answered only by examining the advantages and disadvantages in the individual case.

Among the advantages is, first of all, convenience. The more medications that are taken concurrently and the more complicated the directions, the less likely the patient is to take medications accurately. From the standpoint of convenience and accuracy, and economy as well, you can make an important case for putting medications together in one preparation, as long as they are compatible.

By the same token, when you prescribe a properly tested and rational combination, you should have less worry about pharmaceutical or pharmacological compatibility – and about reasonable dosage ratios as well. Compatibility of the formulation should be demonstrated in the laboratory and clinic before the product is available for prescription–which is more than can usually be said for the physician's own spontaneous creations. And, the dosage ratios employed in rational precompounded combinations are designed to meet the needs of substantial numbers of "typical" patients.

There is no doubt that many "atypical" patients are to be found, and for them the prefabricated combination must be rejected. But that hardly argues for eliminating rational combinations from the market. Think, for example, of the problems that would arise if the components of widely accepted combinations, like the oral contraceptives and the diuretic-antihypertensives, always had to be prescribed, purchased and ingested separately.

One disadvantage that comes to mind is some doctors' unawareness of the ingredients a given combination contains. For example, a doctor might know that a patient is allergic to aspirin but forget that a certain analgesic mixture, which he knows only by its trade name, contains aspirin. His prescription, then, causes considerable discomfort, to say the least. This problem is a function of physician education, rather than of combination therapy as such. Improving doctors' knowledge about all medicaments they prescribe is a problem that deserves tackling on its own.

Another accusation leveled at combination drugs is that they encourage sloppiness of diagnosis and treatment. In many cases, however, a combination may prove to be the most effective choice. A good example of the usefulness of combinations appears in a recent article in the *Journal of Chronic Diseases* on the efficacy and side effects of an antihypertensive containing three ingredients, in which the track records of the combination drug and the individual ingredients were compared. Interestingly enough, whether the drugs were given individually or together, incidence and severity of side effects were the same. But blood pressure control was invariably better when the drugs were taken in one combination tablet than when they were taken separately (in "titratable" dosage) or in two or three different tablets.

Deciding which combinations constitute rational therapy obviously leads to a discussion of who is to determine which should be used and which should not. Realistically, I think combinations should be evaluated somewhat differently if they are old and established or new and untried.

In today's regulatory atmosphere, there is no possibility of a new combination being put on the market without a substantial amount of acceptable evidence in the form of controlled trials that show it to be safe and efficacious. On the other hand, I believe a different set of standards should apply to combination preparations that have been around for a long time. In other words, physician acceptance over a long period should be given some weight as evidence of the efficacy and safety of these drugs.

The FDA, however, does not seem to share this attitude. It often requires, for these older products, controlled trials that will monopolize the time of already overtired investigators and cost a great d of money. I wish we cou agree on a "grandfath clause" approach to pre rations that have been in u for a number of years a that have an apparen satisfactory track record

For example, I thi some of the antibiotic co binations that were tak off the market by the FI performed quite well. I thinking particularly penicillin - streptomy combinations that patie –especially surgical tients–were given in injection. This made less discomfort for the tient, less demand nurses' time, and few opportunities for dosa errors. To take sucl preparation off the mar doesn't seem to be g medicine, unless actual age showed a great deal harm from the injecti (rather than the pro use) of the combination

The point that should emphasized is that th are both rational and ir tional combinations. T real question is, who sho determine which is whi Obviously, the FDA m play a major role in m ing this determination. fact, I don't think it avoid taking the ultim responsibility, but it sho enlist the help of outs physicians and experts assessing the evidence in making the ultimate cision.

laker of Medicine

Clarke Wescoe, M.D.
President
nthrop Laboratories

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sm generated against combinations generally. Obviously, no one should be exposed willy-nilly to the potential side effects of two or three antibiotics when only one is needed. At the same time there are cases where it is prudent to prescribe more than one. The clinician is the judge in these circumstances, as he should be.

There is no clear definition of the word rational. Most persons, I suppose, would find it synonymous with reasonable, but in many circumstances it may best be defined as the opinion of those in power at the moment.

Other factors govern combination therapy, not the least of which has been its broad use by practicing physicians anxious to achieve convenience in prescribing, to reduce medication error, and to save money for their patients. Combinations meet the test on all three counts.

I have been impressed by studies showing that the rate of error climbs markedly with the number of medications to be taken, even with sophisticated patients. When medically justified, therefore, this factor alone supports the logic of combination therapy.

The cost argument for combinations appears to be irrefutable. In 1971, R. A. Gosselin studied the 71 combination products (excluding oral contraceptives) among the 200 most prescribed drugs. The study found that if all 71 products were discontinued, and if each ingredient in these combinations were prescribed separately, the price of medicines to patients would jump by $443.2 million on a national basis! At a time when the cost of medical care is under so much fire, it would be nonsensical to boost costs without clearly irrefutable medical reasons.

The part played by government on this question, of course, is fundamental. The FDA should play a role in determining which combinations are reasonable. That role, as defined by law and regulation, is to ensure that any medication on the market is safe and effective in line with its label claims. Certainly combinations are entitled to as much consideration as single entities—neither more nor less. So long as the addition of one drug to another does not make either less safe, or less effective, so long as they are compatible in a formulation, we have a reasonable product. It makes no sense to recommend the use of two products for certain conditions and to deny their being combined in a single form. An unhappy side effect of the problem concerns the efficacy panel discussions of many products submitted for review. The term "effective, but" has been freely interpreted to mean "ineffective" in toto, regardless of the merit of the individual drugs. This interpretation has placed numerous useful combination products in needless jeopardy.

In reading the actual reports of the review panels, it seems clear that some of the ratings were based less on scientific research and clinical observation than on the "informed" opinions of the panelists. These "informed" opinions were accepted at face value, while the "informed" opinions of others who had used the products were rejected. All of this put combination products into a sort of scientific never-never land.

It should be kept in mind by all, government as well as others involved in our health care system, that advances in therapy are seldom made in leaps and bounds but rather by small painstaking steps—and that some of these steps have resulted from research in combination drugs as well as with single entities. Given the near-infinite biologic variation in patient response, this is hardly surprising to clinicians. It should not be to regulatory agencies either.

In the end, the practicing physician is in the best position to decide if a particular combination makes sense. Such a decision should not be made exclusively by those whose responsibility for continuing clinical care is limited. Clinicians are the best judges of efficacy because the ultimate proof of any product's effectiveness is acceptance by physicians who have observed its actions in patients over time. The corollary statement may be made about over-the-counter medicines, which would not long survive if they failed to afford the relief the user anticipates. That the antihistamine in a "cold" remedy may not *always* be necessary is no reason to proscribe the combination generally.

Opinion & Dialogue

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Evaluation of a Commercially Available Carbohydrate Solution for Oral Use in Glucose Tolerance Testing*

WALTER V. R. VIEWEG, CDR MC USN AND RICHARD E. REITZ, M.D.

Complex carbohydrate solutions have gained wide acceptance for testing glucose tolerance orally because of their commercial availability, improved palatability, virtual absence of side-effects, and ease of storage and handling. However, as recently as 1968 the American Diabetes Association recommended that only preparations containing pure glucose be utilized for testing glucose tolerance.[1] The concern was that solutions containing disaccharides and/or polysaccharides "should be avoided because of possible differences in their influence on intestinal absorption rates." Two studies[2,3] testing a total of 94 patients showed that 75 to 100 gm of a commercially available carbohydrate solution† compared favorably to a solution containing 100 gm of pure glucose when blood glucose levels were measured over a two-hour period in both diabetic and nondiabetic patients. This study was initiated in order to determine whether there is delayed intestinal absorption of a complex polysaccharide solution during a five-hour period after ingestion.

Materials and Methods

Twenty-five young healthy men, aged 18-36, participated in the study (tables 1-3). There was no history of diabetes mellitus in any of the subjects. No medications were administered during the study. Most subjects had recent minor surgical procedures, including inguinal hernia repair and pilonidal cystectomy. No wounds were infected, and all patients were afebrile. All volunteers were given a diet containing at least 300 gm of carbohydrate per day for a minimum of seven days prior to testing. Carbohydrate tolerance tests were performed in the post-absorptive state. The two solutions used were a commercially available carbohydrate solution containing 75 gm carbohydrate in 210 cc‡ and 75 gm of pure glucose in 150 cc water flavored with lemon. The carbohydrate solution consisted of a cola-flavored, carbonated, partial hydralysate of corn starch containing the equivalent of 75 gm of glucose. Both testing solutions were consumed over a two-minute period at 8 a.m. The patients remained at bedrest during the entire procedure. Blood samples were drawn from indwelling venous catheters and collected in tubes containing oxalate. Fasting blood samples were obtained and, in addition, at 30, 60, 90, 120, 240 and 300 minutes post-ingestion. Glucose tolerance tests were carried out on consecutive days. Twelve patients received the carbohydrate solution on Day one and glucose on Day two (table 1). Ten patients received glucose initially and the carbohydrate solution on the second day of testing (table 2), and three patients received the carbohydrate solution, glucose, and the carbohydrate solution again on three consecutive days (table 3). Blood glucose was measured by the Technicon Auto Analyzer method.[4]

Results

The results of the oral glucose tolerance tests for each patient using the two solutions are listed in tables 1-3. Fasting samples never showed more than 24 mg/dl absolute difference in plasma glucose values between testing solutions. In 21 of 25 patients the fasting absolute difference was less than 9 mg/dl. At 120 minutes there was never more than 39 mg/dl absolute difference in plasma glucose values between testing solutions, and in 18 of 25 patients it was less than 20 mg/dl. Figure 1 depicts the mean

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The opinions or assertions contained herein are those of the authors and are not construed as official or necessarily reflecting the views of the Cardiology Department of the Navy or the Naval Service at large.

† The bottled product is commercially available as "Glucola," Ames Company, Elkhart, Indiana.

‡ Glucola, Ames Company, Elkhart, Indiana.

TABLE 1: Glucose tolerance test results (mg/dl) in 12 patients receiving 75 grams of a complex carbohydrate solution (C) and then 75 grams of pure glucose (G) on consecutive days

	Time															
Patient	0 min		30 min		60 min		90 min		120 min		180 min		240 min		300 min	
	C	G	C	G	C	G	C	G	C	G	C	G	C	G	C	G
1	101	85	165	118	115	123	103	155	96	124	56	54	67	74	90	76
2	78	79	123	121	144	129	96	88	105	85	54	59	59	64	69	76
3	50	74	78	111	56	83	50	67	52	78	42	67	56	50	50	69
4	94	90	138	142	158	174	88	128	100	88	105	74	79	88	92	92
5	90	94	189	126	175	164	128	135	124	107	59	100	79	82	86	88
6	97	96	135	154	116	123	118	117	115	113	89	114	85	88	85	85
7	98	95	104	156	171	116	149	100	114	110	117	50	98	86	82	86
8	95	103	156	129	150	162	146	137	135	124	96	102	71	80	85	88
9	97	92	115	130	97	80	89	100	85	67	97	91	105	74	85	86
10	100	95	104	128	98	116	88	110	65	104	92	74	98	90	96	94
11	105	102	184	200	209	226	176	213	126	143	60	63	83	72	83	81
12	88	93	124	132	134	146	120	148	116	132	90	82	76	67	90	92
Mean	91	92	135	137	135	137	113	125	103	106	80	78	80	76	83	84
SEM	4.2	2.5	9.7	6.9	12.0	11.8	9.9	10.9	7.2	6.6	7.0	5.9	4.4	3.4	3.5	2.2

TABLE 2: Glucose tolerance test results (mg/dl) in ten patients receiving 75 grams of pure Glucose (G) and then 75 grams of a complex carbohydrate (C) solution on consecutive days

	Time															
Patient	0 min		30 min		60 min		90 min		120 min		180 min		240 min		300 min	
	G	C	G	C	G	C	G	C	G	C	G	C	G	C	G	C
13	92	94	144	140	137	130	146	133	126	135	117	126	74	102	77	74
14	88	84	120	94	103	98	120	120	94	120	79	94	72	64	84	84
15	94	84	149	173	187	187	204	123	120	108	96	50	64	70	79	88
16	79	81	135	129	140	126	122	104	84	94	72	53	72	64	79	74
17	85	79	128	135	122	110	105	119	87	87	79	60	87	81	85	85
18	86	90	150	164	150	164	127	154	111	106	67	67	82	86	94	94
19	92	90	127	145	133	133	97	97	117	90	71	86	79	86	75	88
20	100	94	158	146	125	120	103	117	122	112	97	103	94	83	83	94
21	94	92	128	138	134	126	125	115	129	118	112	72	80	90	90	96
22	82	94	160	168	168	183	156	164	122	137	114	109	71	71	86	90
Mean	89	88	140	143	140	138	131	125	111	111	90	82	78	80	83	87
SEM	2.0	1.8	4.5	7.2	7.5	9.5	10.0	6.6	5.3	5.5	6.1	8.1	2.8	3.9	1.9	2.4

TABLE 3: Glucose tolerance test results (mg/dl) in three patients receiving 75 grams of carbohydrate solution (C), 75 grams of pure glucose (G), and 75 grams of the carbohydrate solution (C) again on three consecutive days

	Time											
	0 min			30 min			60 min			90 min		
Patient	C	G	C	C	G	C	C	G	C	C	G	C
23	97	95	98	114	135	151	130	158	151	127	123	115
24	94	93	89	127	134	114	110	98	111	93	65	102
25	87	87	86	137	164	114	107	122	130	124	102	112
Mean	93	92	91	126	144	126	116	123	131	115	97	110
SEM	2.9	2.4	3.6	6.7	9.4	12.3	7.2	14.4	11.6	10.9	17.0	3.9

	Time											
	120 min			180 min			240 min			300 min		
	C	G	C	C	G	C	C	G	C	C	G	C
23	114	98	125	82	81	100	83	81	83	86	83	85
24	104	89	104	79	95	81	87	77	83	90	104	87
25	102	98	85	82	90	79	82	90	88	93	90	86
Mean	107	95	105	81	89	87	84	84	85	90	92	86
SEM	3.7	3.0	11.6	1.0	4.1	6.7	1.5	3.8	1.7	2.0	6.2	0.6

values of all test results in tables 1-3, comparing the carbohydrate solution to pure glucose. The differences between the two testing solutions were not statistically significant (p = N.S.).

In order to compare the testing solutions in patients with varying degrees of carbohydrate tolerance, the patients were divided into two groups (Fig. 2). Eleven patients in whom all plasma glucose values were less than 161 mg/dl and the 120 minute plasma glucose value less than 121 mg/dl in all tests were placed in one group (Group I), and the remaining 14 patients were placed in another group (Group II). The difference between the two testing solutions in each group was not statistically significant (p = N.S.) (table 4).

West et al[5] in assessing the reproducibility of glucose tolerance tests in 39 patients found that variations in glucose values at 120 minutes commonly ranged between 20 and 40 mg/dl. The variation in plasma glucose values between the carbohydrate solution and pure glucose in our study probably represents the natural variation in glucose tolerance-testing in the same patient rather than a difference between testing solutions.

Discussion

Complex carobohydrate solutions require hydrolysis to simple glucose in the intestinal tract for proper absorption. In the absence of complete or

TABLE 4: Comparison of mean serum glucose levels (mg/dl) after 75 grams of complex carbohydrate solution (C) and 75 grams of pure glucose (G) during oral glucose testing over a five-hour period

Time (minutes)	0		30		60		90		120		180		240		300	
Solution*	C	G	C	G	C	G	C	G	C	G	C	G	C	G	C	G
Group I†																
Mean	87	88	120	129	114	116	101	101	97	92	78	80	80	76	82	84
SEM	3.8	2.3	5.7	3.3	6.8	6.8	5.6	6.2	5.9	4.5	5.3	4.6	4.1	3.5	3.3	2.9
Group II‡																
Mean	93	93	149	147	153	152	130	141	114	118	85	87	82	79	88	86
SEM	1.5	1.7	6.5	5.6	7.4	8.1	6.1	9.1	4.0	3.6	6.2	6.0	2.6	2.4	1.5	1.5

* C = Carbohydrate ("Glucola", Ames Company, Elkhart, Indiana); G = Glucose solution.

† Subjects with dl plasma glucose values less than 161 mg/dl at 60 minutes and 121 mg/dl at 120 minutes.

‡ Subjects excluded by criteria for Group I.

rapid hydrolysis there may be incomplete or delayed absorption. Two studies[2,3] testing both normal and diabetic patients found that the commerically available complex carbohydrate solution tested in this study provided blood glucose levels comparable to pure glucose when tested over two hours. Our studies indicate that the commercially available solution studied compares favorably to pure glucose in oral glucose tolerance-testing over a five hour period. In patients suspected of having a disaccharidase deficiency a delay in absorption of glucose due to impaired hydrolysis to glucose may occur. Further studies in these patients will be necessary to determine whether delayed absorption occurs with these complex polysaccharide solutions.

Summary

A commercially available complex carbohydrate solution was compared to a pure glucose solution in oral glucose tolerance-testing over a five hour period in 25 young men without known diabetes mellitus. The two testing solutions provided essentially identical results. In this, as well as in other reports, results indicate that complex carbohydrate solutions are satisfactory in oral glucose tolerance-testing.

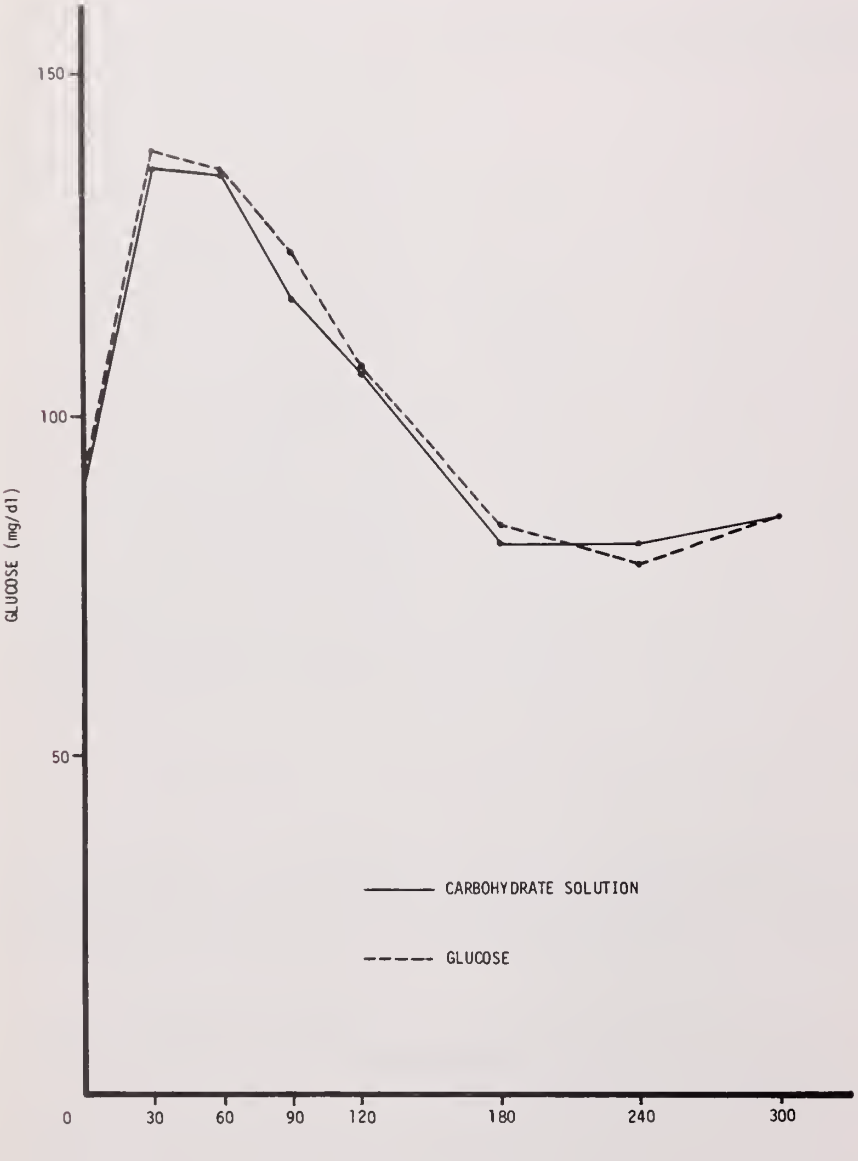

FIG. 1. Comparison of mean serum glucose levels (mg/dl) in 25 adults after 75 gm of a carbohydrate solution and 75 gm of glucose

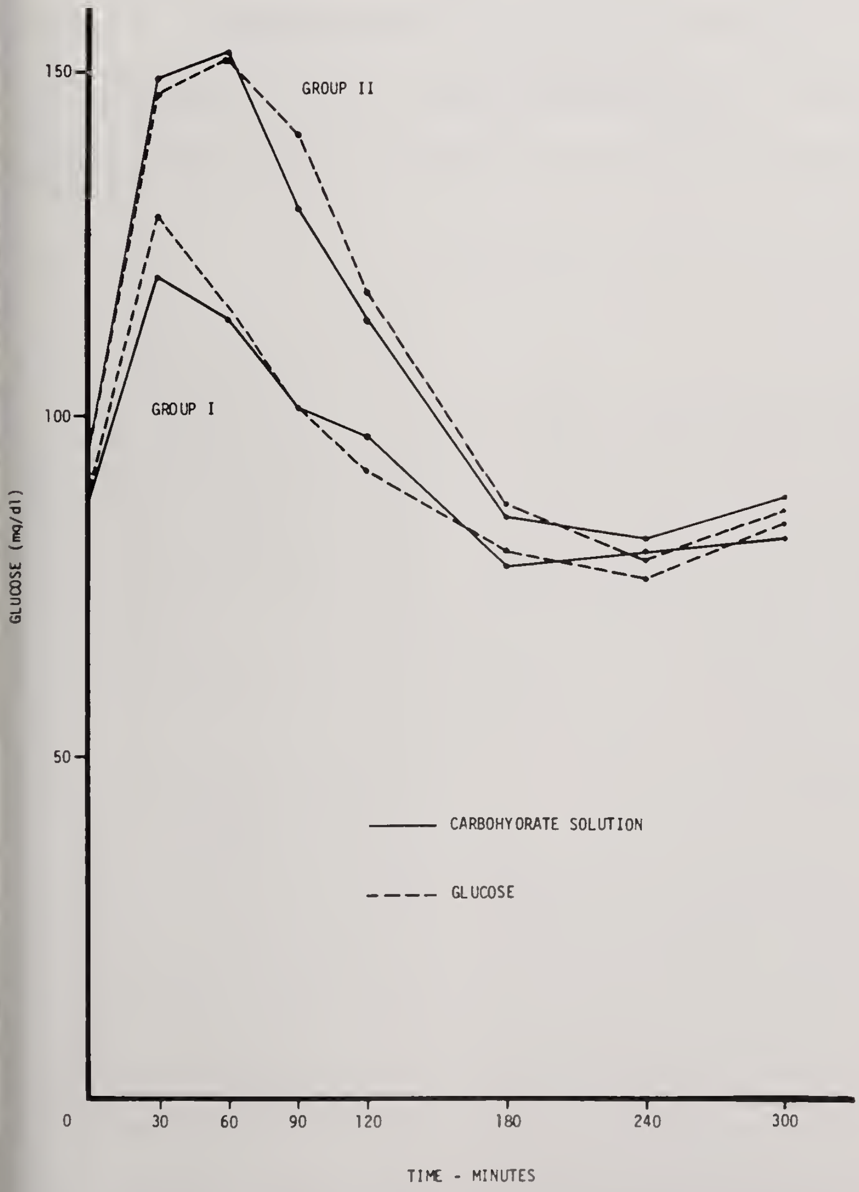

FIG. 2. Comparison of mean serum glucose levels (mg/dl) in Groups I and II after 75 gm of a carbohydrate solution and 75 gm of glucose.

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Hypokalemia with Normal Blood Pressure Preceding Malignant Hypertension in a Patient with Primary Aldosteronism (Conn's Syndrome)[*]

MANUEL T. VELASQUEZ, M.D., NIRMAL SAINI, M.D. AND JAY N. COHN, M.D.

Mild elevation of blood pressure is the common initial feature in patients with primary hyperaldosteronism.[1] Rarely, accelerated hypertension develops as a complication after many months or years of existing benign hypertension.[2,3] Spontaneous hypokalemia in these patients has not been reported before the onset of hypertension.

The following report describes a patient with primary hyperaldosteronism due to adrenal cortical adenoma in whom spontaneous hypokalemia with normal blood pressure preceded by four months the development of malignant hypertension. Removal of the adenoma corrected the hypokalemia and moderated the hypertension.

Report of Case

D. M., a 21-year-old caucasian was referred to Kimborough Army Hospital, Fort Meade, Md. on September 16, 1968 for investigation of mild proteinuria found on a urinalysis performed as part of his Army discharge physical examination. He was asymptomatic except for the recent onset of occasional enuresis. There was a history of hypertension in both his mother's and father's family. The blood pressure was 120/76, and complete physical and ophthalmological examinations were normal. Laboratory data summarized in table 1 were all normal except for a low serum potassium of 2.9 mEq/L. A second urine specimen gave a negative test for protein, and the patient was discharged from the Army.

He remained asymptomatic until January 23, 1969, when he noted painless gross hematuria. He was admitted three days later to the Washington V.A. Hospital with a blood pressure of 200/140. There was papilledema with fresh retinal hemorrhages and exudates. The lungs were clear, and the heart was not enlarged. There was a grade II/VI short systolic ejection murmur and a presystolic gallop at the apex. The abdomen was negative. The electrocardiogram revealed slightly high QRS voltage in the precordial leads but no other abnormalities. The laboratory data are shown in table 1. He was treated initially with intermittent intramuscular injections of hydralazine followed by guanethidine, 30 to 50 mg daily, which maintained his blood pressure at approximately 150/90. A low-salt diet with oral potassium supplements corrected his hypokalemia, and he was then transferred to the metabolic ward.

Method

Biochemical studies were undertaken following a period of at least four days of low-salt (10 mEq) and high-salt (200 mEq) diets. Routine determinations were performed by the Autoanalyzer. Endogenous creatinine clearance was used to estimate glomerular filtration rate. Renin activity of plasma from renal venous and peripheral blood was determined by a bioassay method and is reported as ng angiotensin produced during 4.5 hours of incubation.[4] Aldosterone excretion rates were measured by the double isotope derivative technique[5] by the New England Nuclear Biomedical Laboratory. Urinary 17-ketosteroids and 17-ketogenic steroids were determined by a modification of the method of Few.[6] Plasma aldosterone levels and aldosterone secretory rates were determined by a modification of the method of Kliman and Peterson[5] through the courtesy of Drs. E. Bravo and F. Bartter from the Endocrinology Branch, National Heart and Lung Institute.

Results

The first blood specimens for renin activity were obtained 10 days after his admission with malignant hypertension. Renin activity in plasma obtained from arterial and right and left renal venous blood all ranged from 4 to 6 ng/ml. Subsequently during metabolic balance studies peripheral plasma

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TABLE 1: Summary of Clinical and Laboratory Data

	Initial Examination September 1968	Hospital Admission January 27, 1969	Post-Surgery May to August 1969
Clinical Data			
Symptoms	Enuresis	Gross hematuria and weight loss	Asymptomatic
Blood pressure	120/76	200/140	140/90-150/110
Fundi	Normal	Grade IV	Grade I
Weight (lbs)	165	151	180
Laboratory Data			
Hematocrit (%)	48	47	44
Serum potassium (mEq/L)	2.9	2.2-2.9	4.2-4.8
Serum sodium (mEq/L)	143	142-147	140-147
Bicarbonate (mEq/L)	29.0	29.0-32.0	23.0-27.0
Serum chloride (mEq/L)	100	90-102	95-103
Serum calcium (mg/100 ml)	5.2 (mEq/L)	9.2-9.9	10.2-10.5
Blood urea nitrogen (mg/100 ml)	14	12-18	10-18
Serum creatinine (mg/100 ml)	–	1.2-1.4	1.1-1.4
Total proteins (gm/100 ml)	7.6	6.0-7.2	7.2-8.0
Serum albumin (gm/100 ml)	5.6	4.2-4.5	5.0-5.3
Urine pH	Acid	5.0-6.0	5.0-6.0
Urine protein	Trace to 1+	2+	Negative
Glucose tolerance test	–	Diabetic curve	Normal

TABLE 2: Pre-operative Renin and Aldosterone Values During Low- and High-Salt Diets

	Low Salt	High Salt
Plasma renin activity, ng/ml/4.5 hrs	0	2
Aldosterone excretion, μg/24 hrs	27.0	13.5
Aldosterone secretory rate, μg/24 hrs	–	757

renin activity was negligible during both low- and high-salt diets. Aldosterone excretion, which was abnormally high during salt restriction, fell moderately during the high-salt diet, but the aldosterone secretory rate remained elevated (table 2). The urinary 17-ketosteroids and 17-ketogenic steroids were 10.6 mg per 24 hours (normal 4-23 mg per 24 hours) and 9.1 mg per 24 hours (normal 5-17 mg per 24 hours) respectively.

Endogenous creatinine clearance was 95 ml per minute. Rapid-sequence intravenous pyelography and radioisotope renogram were normal. Transfemoral aortogram showed normal major renal arteries and normal kidney size. Kidney biopsy specimen examined both by EM and light microscopy revealed nephrosclerosis, arteriolar and arterial, and focal nephrocalcinosis.

The serum potassium, which rose from 2.2 to 4.9 mEq/L after oral potassium supplements during salt restriction, fell to 3.4 mEq/L during salt-loading. Potassium excretion increased slightly from 83 to 108 mEq per day (Fig. 1).

Left adrenal vein-blood obtained by retrograde catheterization from the femoral vein contained 13.6 μg aldosterone per 100 ml of plasma. Right adrenal vein-blood could not be obtained.

On April 30, 1969 a 2 cm encapsulated golden yellow tumor was removed from the left adrenal gland. Sections of the adrenal nodule showed epithelial cells with a clear, finely vacuolated cytoplasm and moderately pleomorphic nuclei arranged in cords, strands and nests separated by sinusoids, with slightly thickened walls. Giant nuclei were present. There was no evidence of lymphatic or capsular invasion. A fat stain revealed an abundant amount of lipid throughout the tumor.

Following the operation the patient's blood pressure ranged between 140/90 and 150/110 without medication. The serum potassium ranged between 4.2 and 4.8 mEq/L while on a regular diet. Clinical and laboratory data are summarized in table 1. In contrast to pre-operative responses to changes in salt intake, serum potassium and potassium excretion did not change in response to salt-loading in the postoperative period. Plasma renin activity was in the normal range

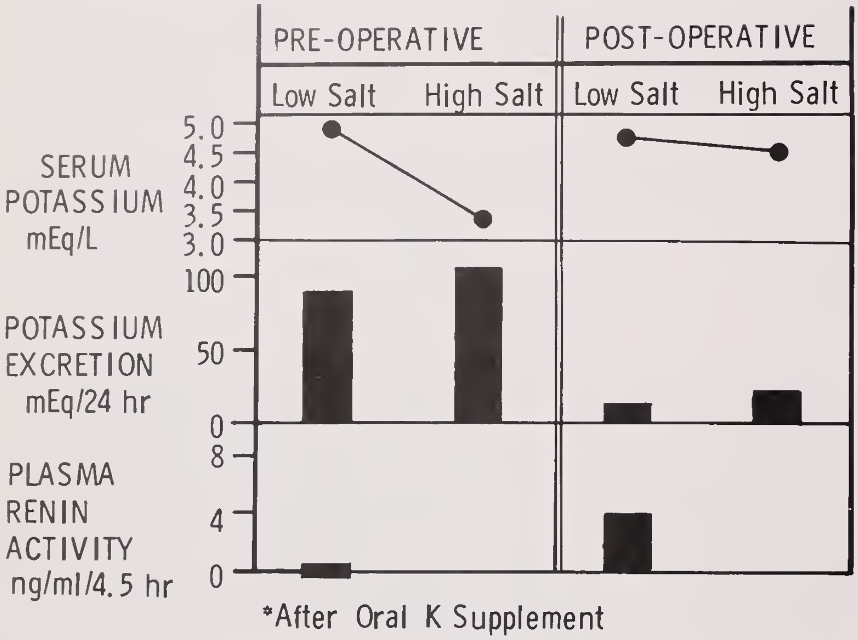

FIG. 1. Electrolyte and renin assay values before and after removal of adrenal cortical tumor.

following surgery. The enuresis which had continued to be troublesome during hospitalization disappeared following surgery. For two years after surgery the diastolic blood pressure ranged between 90-110 mmHg without therapy. The patient was then begun on therapy with a thiazide diuretic which has normalized the blood pressure without inducing hypokalemia.

Discussion

Two aspects of the course of this patient with primary aldosteronism are unique. One was the spontaneous hypokalemia prior to the onset of hypertension and the other was the development of malignant hypertension without preceeding chronic benign hypertension. The serum potassium of 2.9 mEq/L noted four months prior to admission was not further evaluated at the time, but subsequent questioning of the patient revealed no gastrointestinal disturbances or unusual diet or drug ingestion which could have been responsible.

It is not known when in the four-month interval between blood pressure recordings that hypertension developed. However, the normal optic fundi noted at the earlier examination and the normal chest X-ray film and the ECG at the time of the later hospital admission when there was grade IV fundi indicates that the course of the hypertensive disease was telescoped. No explanation can be offered for the unusually severe and accelerated hypertension, except that perhaps the primary aldosteronism was superimposed on a "pre-hypertensive" state. Indeed, the persistent postoperative mild hypertension may be viewed as a manifestation of early essential hypertension with a strong family history of hypertension.

Secondary hyperaldosteronism as a cause of the hypokalemia probably can be ruled out. The peripheral renin activity was not elevated, and the patient exhibited no signs of hepatic or cardiac disease. Renovascular hypertension was excluded by a normal renal arteriogram and bilaterally low and equal renal venous renin-activity during salt restriction. Furthermore, the renal biopsy showed no evidence of pyelonephritis or glomerulonephritis to support a diagnosis of salt-losing renal disease. Although hypokalemia and normal blood pressure have recently been reported by Fichman et al[7] as a manifestation of renal-cell carcinoma and adrenal cortical adenoma, their patient exhibited normal renin and aldosterone levels. The extremely high aldosterone concentration in left adrenal vein-blood and the typical histology of the adenoma are strong evidence that this tumor was the source of the metabolic abnormality. Correction of the hypokalemia and hyporeninemia after removal of the tumor provide the final proof of the diagnosis.

The failure to suppress aldosterone secretion or excretion during salt-loading has been stressed by some investigators[8-10] as an important additional tool in differentiating primary aldosteronism due to adrenal cortical adenoma from secondary hyperaldosteronism. However, this was not the case in the present study since aldosterone excretion was substantially suppressed following a high-salt diet. This suppression, which may be due to the inhibitory effect of potassium depletion on aldosterone secretion,[11] strengthens the suggestion of George et al[10] that some degree of normal physiological control of aldosterone secretion still is operating in patients with an aldosterone-producing adenoma.

It is suggested that hypokalemia may precede the development of hypertension in patients with primary aldosteronism due to adrenal cortical adenoma. These data are at variance with the reports of Conn,[12,13] who

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has suggested that hypertension precedes the onset of hypokalemia and that normokalemic hypertension may in many patients represent an early stage of primary aldosteronism. Whether the sequence in the present case is an unusual presentation of the disease or a common occurrence not previously reported because electrolyte-concentrations usually are not measured in normotensive patients must at the moment remain conjectural.

Summary

Spontaneous hypokalemia with a normal blood pressure was noted 4 months prior to the onset of accelerated hypertension in a 21-year-old man with an aldosterone-secreting adrenal cortical adenoma. Suppressed renin activity despite malignant hypertension associated with a typical biochemical response to salt-loading and salt-restriction confirmed the impression of Conn's syndrome. Removal of the adrenal adenoma was accompanied by moderation of the hypertension and correction of the kypokalemia and hyporeninemia. The chance finding of hypokalemia prior to hypertension in this patient indicates that hypertension may not be the earliest manifestation of primary aldosteronism.

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Metastatic Meningeal Carcinomatosis. Report of a Case and Review of the Literature

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About 5% of patients with carcinoma develop cerebral metastases.[1] These are usually nodular, sharply circumscribed, and distributed according to the volume of the various parts of the cerebral tissue. Spread of tumor cells diffusely into the subarachnoid space is uncommon and occurs with primary intracranial tumors, especially medulloblastomas and, less often, glioblastomas.[2]

In these cases, however, there is a grossly obvious lesion from which the tumor cells have spread. Least common of the metastatic lesions is the diffuse spread of tumor cells in the pia-arachnoid without detectable involvement of the cerebral tissue. In such cases the meninges often appear grossly normal, and the cut surfaces of the brain fail to reveal metastatic disease. Cases of this kind are morphologically remarkable and raise certain questions. For example, what patients are affected, what clinical manifestations arise, and what tumors spread to the coverings of the brain in this remarkable way? What methods are available for the diagnosis of this unusual condition? In order to present this entity and answer these questions, the following case is described.

Report of Case

The patient was a black man 70 years old who was admitted to the hospital because of a change in mental status. He had been well until 1 month previously when the family noted confusion, visual illusions, and instability on walking. These persisted, and two weeks before admission buzzing developed in both ears. The difficulty in walking increased. Two days before admission the patient experienced three episodes of nausea and vomiting. He had smoked two cigars daily for many years but did not smoke cigarettes. During the history taking, the patient had periods of confusion, momentarily forgetting where he was, and at another time mistaking a television screen for a goldfish bowl.

The vital signs were normal. Physical examination was not remarkable except for the neurologic findings. These included staggering to the right on walking and falling forward during the Romberg test. Coordination was diminished, especially on the left. The deep tendon reflexes were active and equal. The Babinski reflex was negative on both sides.

The hematocrit was 43%, the leukocytes numbered 7,200, and the differential count was normal. The blood urea nitrogen, serum calcium, total bilirubin, serum glutamic transaminase and lactic dehydrogenase were normal. Lumbar puncture revealed clear fluid with 17 lymphocytes per cmm. The protein and sugar were 57 and 42 mg. per 100 ml., respectively, and the opening and closing pressures were normal. A chest X-ray film disclosed blunting of the costophrenic angles, hilar calcifications, and a hazy infiltrate at the right apex. Skull X-ray films revealed a midline calcified falx. An electroencelphalogram was diffusely abnormal, and a brain scan was interpreted as within normal limits. A diagnosis of tuberculous meningitis was considered, and the patient was given streptomycin, aminosalicylic acid, and isoniazid. His condition remained the same for the next three days, and on the fourth hospital day the lumbar puncture was repeated. The results were unchanged.

On the ninth hospital day, a right brachial-carotid arteriogram was interpreted as normal. The lumbar puncture was repeated, and the results were unchanged. On the next day neurological examination revealed deterioration but no localizing physical signs. Smears and cultures of sputum and spinal fluid for pyogenic and acid-fast organisms were negative. Biopsies of liver and bone marrow were normal. The patient lapsed into coma on the 15th hospital day and died two days later.

Autopsy examination revealed a primary carcinoma of the lung with metastatic carcinoma of the cerebrum, cerebellum, and leptomeninges. No

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malignant tumor was found outside of organs, i.e., lungs and brain. Additional diagnoses included chronic pulmonary emphysema, a cortical adenoma of the right adrenal, and nodular hyperplasia of the prostate.

The pulmonary cancer was bronchoalveolar in type, and was present in small separate foci widely scattered throughout both lungs. These were barely discernible on the pulmonary cut surfaces. The precise site of origin in the pulmonary tissue was thus not determined. Histologic examination revealed columnar cells in single layers that tended to line the alveoli without destroying the tissue pattern. A microsection of the pulmonary tumor is shown in figure 1.

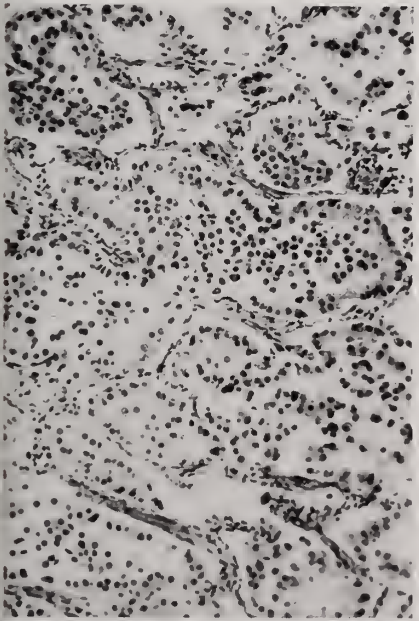

FIG. 1. Microsection of lung showing alveolar-cell carcinoma lining the air sacs.

The brain weighed 1600 gm. The cerebral hemispheres were symmetrical, and the meninges were transparent except for a small number of minute white flecks on the undersurface. After several days fixation in formalin, however, re-examination disclosed a pale gray slight translucency of the leptomeninges over the entire cerebellum, the inferior aspects of the temporal lobes of the cerebrum, the medial aspects of the occipital lobes, and to a lesser extent the region of the interpeduncular cistern. Although no thickening was evident, this discoloration tended to obscure the subjacent vessels and sulci. There were also scattered minute pinpoint nodules in the leptomeninges over portions of the cerebellum. The superior and inferior surfaces of the brain are shown in figures 2 and 3.

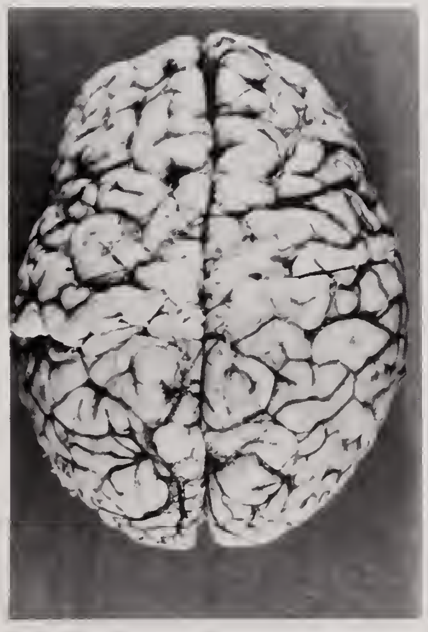

FIG. 2. Superior surface of cerebrum with normal-appearing meninges.

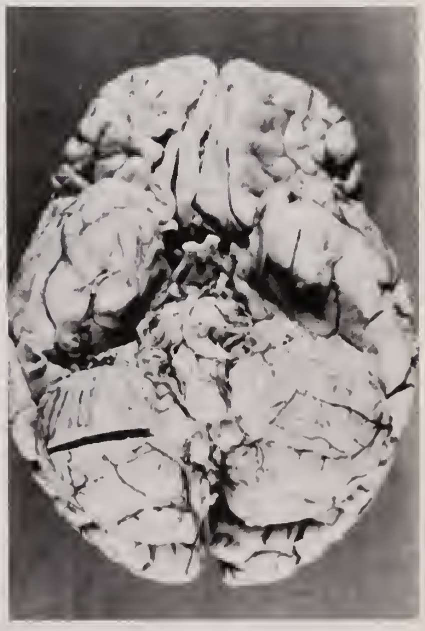

FIG. 3. Inferior surface of brain with normal-appearing meninges.

Coronal sections of the brain at 0.5 cm increments revealed four soft, pale gray nodules measuring 1.0 cm in greatest dimension that were scattered within the cortex in haphazard distribution. Also present in the depths of the sulci were poorly demarcated foci of softening that extended into the cortex and were irregularly distributed over the cerebrum. The cut surfaces of the cerebellum revealed patchy foci of pale gray discoloration measuring up to 1.5 cm in greatest dimension that focally obscured the foliate structure of that tissue. The cut surfaces of the brain stem were not remarkable. The cerebral ventricles were symmetrical, the ependymal surfaces were smooth, and the choroid plexus was not unusual. The spinal cord was not removed.

Histologic examination revealed metastatic tumor throughout the leptomeninges of the cerebellum and virtually all of the cerebrum. The tumor cells showed a tendency to grow along capillaries and small blood vessels with regular extension into the bases of the sulci, and infrequent extension into the subjacent cortical tissue via the Virchow-Robins perivascular spaces. The localization of the tumor cells in the subarachnoid space is shown in figure 4. Atrophy of the cortical granular and molecular layers was present at many sites. Sections of brain stem also revealed extensive growth of tumor within the subarachnoid space, the tumor cells extending around and within venules. The pineal gland was invested in tumor with marginal invasion of the structure. Ischemic necrosis was abundantly evident in the cortical neurons of the cerebrum, the Purkinje cells of the cerebellum, and the pyramidal neurons of the hippocampus. No tumor was found outide of the leptomeninges and the cortex of the cerebrum and cerebellum. The morphologic diagnoses thus included diffuse metastatic meningeal carcinomatosis and widespread hypoxic encephalopathy of the central nervous system tissue.

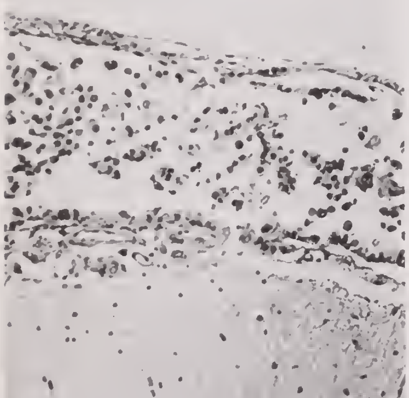

FIG. 4. Microsection of cerebral surface showing diffuse spread of tumor cells within the pia-arachnoid.

Comment. Noteworthy observations include the extensive distribution of tumor within the meninges, the multiple foci of primary carcinoma within the lung, and the absence of metastases in any other organ. Also noteworthy is the essentially normal cerebrospinal fluid which differs from the usual finding in meningeal carcinomatosis.

Discussion

According to Dinsdale and Taghavy, the first case of diffuse metastatic meningeal carcinomatosis was described by Ollivier[3] in 1837. Fischer-Williams and colleagues,[4] however, attribute the first case to Eberth in 1870, although he failed to associate the co-existence of a carcinoma of the lung with tumor cells in the leptomeninges. Thereafter, several cases were reported in the German literature, and the terms "metastatic meningitis," or "meningitis carcinomatosa" were widely used. An early case appearing in the English literature was recorded by Beerman[5] in 1912, who used the more nearly correct term, "meningeal carcinomatosis." Since then several reviews of the condition have appeared; especially noteworthy are those of Jacobs and Richland,[6] Grain and Karr,[7] Dinsdale and Taghavy,[3] and Fischer-Williams, Bosanquet, and Daniel.[4]

The incidence of metastatic meningeal carcinomatosis is not altogether clear; some authors[8,9] regard it as rare, while others describe it as "uncommon but not rare."[3] Madow and Alpers[10] found four cases among 106 patients in whom there were cerebral metastases. In 1951 Jacobs and Richland[6] found 55 cases in the literature, Strange[11] described 18 cases of her own observed over a 20-year period, and Grain and Karr[7] were able to tabulate 85 cases by 1955. Takahashi[12] stated that about 120 cases had been reported in Japan to 1970. Nevertheless, Fischer-Williams and colleagues[4] pointed out that the incidence of the condition is unknown because of differing definitions of it, and Heathfield and Williams[13] emphasized that cases may be missed because of failure to make histologic sections of brains that appear grossly normal.

Most authors disqualify cases in which a cerebral metastasis is grossly noticeable, and restrict the entity to those in which the cut surfaces of the cerebrum and cerebellum appear altogether normal.[6,8,3] Some, however, accept cases in which the leptomeninges are affected "more or less exclusively."[14] It appears that all gradations exist from grossly evident cerebral metastases with moderate leptomeningeal spread, to diffuse involvement of the meninges without identifiable metastases in the cerebral tissue. Most cases lie between these extremes, with extensive spread within the pia-arachnoid, and metastatic tumor in the cerebral cortex that is identifiable only by histologic examination. In some cases the meningeal metastases are localized to the spinal cord.[11,15]

In the reported cases, with metastatic disease affecting the meninges "more or less exclusively," the patients have been adults, somewhat more often male than female. The age and sex distribution is determined mainly by the primary tumors from which most cases arise, i.e., carcinomas of the breast exclusively in the female, and carcinomas of the lungs and stomach in both sexes although more often in the male. The age range, as reported by Herman and Courville[14] ranged from 20 to 84 years. The range of the 55 cases noted by Jacobs and Richland[6] was 20 to 66 years, and the average age was 45.5 years. The 18 patients described by Strange[11] were between 48 and 60 years of age. In most reports, the frequency is greater in males than females, e.g., 74 males to 62 females,[14] 30 males to 20 females,[6] 52 males to 27 females,[7] and 15 males to three females.[11] There were, however, nine females and four males in the cases reported by Takeuchi and Yahagi.[9]

Morphologic Features

The most common gross finding on examination of the brain is a slight gray haziness of the meninges.[8] This is easily missed, however, and in many cases no abnormality is seen, i.e., the lobes are symmetrical, the ventricular system is not dilated, and the ependymal surfaces are smooth and glistening. Moreover, the cut surfaces are not remarkable, with intact cortical gray matter and uninvolved subjacent white matter. Histologic sections, however, reveal metastatic tumor throughout the subarachnoid space of the cerebrum, cerebellum, and often the spinal cord. The tumor dips into the sulci and may extend within perivascular spaces into the superficial layers of the cerebral cortex. In a report of 18 cases[11] tumor cells were found only in the meninges in eight and in the meninges and subjacent cortical tissue in 10. The base of the brain tends to be more affected than the superior surface,[8] and the posterior surface of the spinal cord more than the anterior.[16] The cranial nerves, especially the optic tracts, are often involved, as well as the spinal nerves, particularly in the lumbar and sacral regions. Tumor cells around spinal nerves were found in seven of 18 cases in one report,[11] and in 17 of 85 cases in another.[7] The ventricular system is unusually unobstructed, although in a few cases tumor cells are found on the ventricular surfaces without infiltration of the ependyma. The pineal and choroid plexus are usually not involved.

The primary carcinomas from which most cases arise are in the stomach, lung and breast. Other sites, however, also give rise to such metastases, and the number of cases according to the sites of origin indicates the frequency of each. In the 147 cases tabulated by Herman and Courville,[14] the primary sites were stomach 53, lung 36, breast 16, others 22, and undetermined 20. The same data for the 85 cases noted by Grain and Karr included stomach 33, lung 22, breast 12, others 14, and undetermined 4. Jacobs and Richland[6] made a similar analysis; of 52 cases, 23 arose in the stomach, nine in the lung, six in the breast, four elsewhere, and in 10 the primary site was undetermined. The "other" primary sites include, esophagus, pancreas, gallbladder, cecum, colon, rectum, adrenal gland, skin (melanoblastoma) prostate, and testis.[14] Takeuchi and Yahagi[12] reported a single case arising in the thyroid. Carcinomas of the kidney and thyroid, which frequently metastasize to the brain, are conspicuously absent from the list of tumors associated with diffuse meningeal carcinomatosis.

Carcinoma of the stomach rarely metastasizes to the brain; only three examples were observed by Herman and Courville in over 75,000 autopsy examinations. Nevertheless, the stomach is the most common primary site for metastatic carcinomatosis of the meninges.[9,7,6] This observation is also noted elsewhere; in Japan gastric carcinoma is extremely common but rarely metastasizes to the brain. Regardless of this, more than half of the 92 cases reviewed by Takahashi arose in the stomach. He thus points out that when gastric cancer metastasizes to the brain it is more likely to spread into the meninges in diffuse form than into the cerebral tissue in nodular form. With respect to lung, most primary malignant tumors of that organ are squamous carcinomas, and most cerebral metastases from such tumors are nodular and within the cerebral tissue. It is the adenocarcinoma of lung, however, that most often gives rise to diffuse meningeal metastases even though such pulmonary tumors show little tendency to metastasize to the brain. In the observations of Decker,[17] for example, only five of 155 adenocarcinomas of the lung revealed cerebral metastases.

Microscopically, most of the meningeal metastases are adenocarcinomas. Of the 92 cases reviewed by Takahashi, for example, 82% were gland-bearing in histologic type. This is because of the glandular nature of the stomach and breast and the special tendency for adenocarcinomas of the lung to spread in this way. Hence an affinity of gland-bearing tumors for the meninges is suggested. It is also observed that most of the metastases are well differentiated so that the degree of malignancy does not appear to be an important determinant of the meningeal localization. In addition, in many cases few or no metastases are found except in the meninges, and it is apparent that if anaplasia were a determinant of meningeal spread, extensive extracranial metastases would also be expected.[3,7,16,18] In the case herein described, only meningeal metastases were observed.

Much consideration has been given to the route by which the tumor cells reach the subarachnoid space. The possibilities given most attention are the arteries, the vertebral veins of Batson, and the perineural lymphatics. Also mentioned are seeding of the cerebral ventricles from secondary deposits within the choroid plexus and seeding of the subarachnoid space from cortical metastases in the cerebrum. While there is no agreement on these possibilities, Lesse and Netsky[1] favor the arterial route, and Fischer-Williams and colleagues[4] concur in this opinion. Others, however, favor a venous route, pointing out that direct connections link the veins of the thorax and abdomen with the epidural veins of the vertebrae.[6] Metastasis through the Batson system of veins also appealed to Takahashi.[12] Several have spoken against this route, however, and Herman and Courville[14] pointed out that the infrequent involvement of the vertebral bone marrow militates against this possibility. Other evidence has been credited to the perineural lymph route; Takahashi. thought it most likely, Grain and Karr observed it in a case of gastric carcinoma spreading diffusely to the meninges, and Fischer-Williams and colleagues noted perineural infiltration in two of their three cases. Alpers and Smith[16] supposed a perineural route in their case of lung cancer in which the meningeal metastases were limited to the spinal cord. In addition, Herman and Courville cited "extensive spread from the primary tumor [cecum] into the spinal canal by way of the lower spinal nerves". Although Jacobs and Richland pointed out that the perineural spaces are not clearly lymphatic, they nevertheless accepted the plausibility of this route because of the passage of the spinal nerves through the coverings of the cord before connecting with that tissue. In two of the three cases of Dinsdale and Taghavy, the choroid plexus was involved, and they suggested seeding from that site into the ventricular system. While that possibility exists, involvement of the choroid plexus is infrequent,[7,8,19] and meningeal spread is more pronounced over the base of the brain than around the foramina of the fourth ventricle.[16]

In the cerebral cortex, tumor cells are often observed within the perivascular spaces of Virchow-Robin, and it is presumed that these cells are invading the cortical tissue. This interpretation is based on the finding of abundant tumor in the subarachnoid space and scant tumor within the cerebral cortex. Nevertheless, the possibility exists that a cortical metastasis is seeding the leptomeninges since cortical involvement is not infrequent although often only microscopic. This possibility has been pointed out by Fischer-Williams and colleagues and by Grain and Karr. Moreover, this mechanism is evidenced by the case of Plaut[20] in which a paranasal carcinoma eroded into the subarachnoid space and spread diffusely in the meninges of the brain and spinal cord. In addition, in the case of DeVita and Canellos[21] a single nodule of melanoblastoma in the parietal lobe of the cerebrum was associated with diffuse seeding of the meninges with tumor cells. Thus, seeding of the subarachnoid space from an inconspicuous cortical nodule could also account for this unusual distribution of metastatic tumor.

Clinical Aspects

The clinical manifestations of metastatic meningeal carcinomatosis are so complex as to make the diagnosis "very difficult."[14] Indeed, Takeuchi and Yahagi[9] declared that there is "no recognizable clinical picture." However, others have found a recurring pattern, and in two of the cases reported by Strange[11] the correct diagnosis was made. The clinical pattern consists of mental changes, meningeal signs, and cranial nerve palsies associated with unusual spinal fluid. The mental changes include some combination of memory loss, disorientation, confusion, restlessness, stupor, confabulation, psychotic episodes, hallucinations, convulsions, and speech and auditory disturbances. There may also be nausea and vomiting, back or radicular pain, sensory or motor loss, and fecal or urinary incontinence. Meningeal signs include headache, which is usually early and often severe, and nuchal rigidity which is often early but may come on late. Paresis of several cranial nerves is the most consistent clinical finding according to Fischer-Williams and colleagues. Chief among these are oculomotor and facial nerve palsies associated with visual impairment which frequently advances to blindness. The visual disturbance is often accompanied by unequal pupils and papilledema. Over one-third of the patients reviewed by Fischer-Williams and colleagues became blind in the course of their disease. The patients are regularly afebrile. Thus the clinical diagnosis is suggested by a middle-aged patient of either sex, with or without a known primary carcinoma, who develops a severe headache, progressive mental changes, often meningeal signs, and visual impairment that relentlessly worsens. These manifestations are attributed by Dinsdale and Taghavy to interference with cerebral metabolism since inflammation is absent from the meninges and tumor-cell infiltration of the brain is scant.

The cerebrospinal fluid is clear, sterile, and often only slightly abnormal. The abnormalities include an elevation of pressure which is occasionally pronounced, a slight pleocytosis, and a moderate increase in protein. There is, however, both a characteristic feature and a diagnostic one. The characteristic feature is a low spinal fluid glucose.[22] In their review of 58 cases, Kilpatrick and Pankey[23] found a low sugar level in the spinal fluid of 45 (78%). This finding has been attributed to increased utilization of glucose by the tumor cells[18,24] and to an impairment of glucose transfer across the blood-brain barrier.[13] Evidence of the latter has been provided by Fishman[25] who was unable to raise the glucose level in the spinal fluid by injecting sugar intravenously in two patients with diffuse meningeal carcinomatosis. The matter is of some importance, as pointed out by DeVita and Canellos[21] because of the possibility that therapeutic agents given intravenously may not be able to cross the blood-brain barrier. Completeness requires mention of the atypical case of Tegeris and Brandriss[26] in which a solitary hemangioma of the cerebellum without diffuse meningeal spread was associated with a low cerebrospinal fluid sugar-level. Spinal fluid of this character is suggestive of tuberculous meningitis, torulosis, and sarcoidosis.[23] Of these, the first is the most likely, and in several cases, including ours, antituberculous therapy was given on empirical grounds.[21,24,26] The fluid is, however, sterile.

The feature that establishes the diagnosis is the finding of tumor cells in the spinal fluid. This is frequently done, and McCormack and colleagues[19] have recommended a stain that has been "extremely satisfactory" in their hands. Occasionally signet-ring cells giving a positive mucicarmine stain are identified.[8] Other general laboratory findings tend to be normal, and angiographic, electroencephalographic, pneumo-encephalographic, and ventriculographic findings are often not remarkable or are nonspecific. It is concluded that visual impairment and severe headache together with mental changes and multiple nerve palsies in an afebrile middle-aged patient suggest the diagnosis whether or not a primary carcinoma is clinically evident. The diagnosis is supported by the finding of a sterile spinal fluid in which the glucose level is low and is confirmed by the finding of malignant tumor cells in the fluid.

After the onset of neurologic symptoms, the progress of the disease is relentless and rapid. Coma ordinarily precedes death. In various reviews the patients succumbed within two weeks to four months,[9] in less than three months,[1,7] and in less than four months.[4] The average duration according to Dinsdale and Taghavy[3] was six weeks, and in the 92 cases tabulated by Takahashi[12] was two months. While therapy has often been unapplied because of failure to recognize the condition, Heathfield and Williams[13] achieved a two-year survival and recovery from blindless by whole-brain irradiation. Antimetabolite therapy, however, has not been successful, including the use of intrathecal amethopterin. Thus, amelioration of symptoms appears to be the most that can be achieved, and irradiation seems to be the modality most likely to accomplish that.

Summary

Diffuse meningeal carcinomatosis is the least common form of intracranial metastasis. The tumors are usually well differentiated adenocarcinomas that arise in the stomach, lung, or breast. The tumor cells may reach the meninges by hematogenous dissemination, retrograde perineural lymph flow, or by seeding from an inconspicuous metastasis in the cerebral cortex. The patients manifest mental and meningeal signs associated with multiple cranial-nerve palsies, especially visual impairment which may progress to blindness. The spinal fluid is characterized by an increase in pressure and protein, a decrease in sugar, and a stained sediment in which tumor cells are identifiable. Amelioration of symptoms and prolongation of life have been achieved only by whole-brain irradiation. An additional case has been reported herein.

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Complete Heart Block Occurring During Right-Heart Catheterization in a Patient with Left Bundle-Branch-Block and Prolonged P-R Interval Studied by Bundle of His Recording and Atrial Pacing*

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Right bundle-branch-block is a recognized complication of right-heart catheterization.[1] If right bundle-branch-block occurs during cardiac catheterization in a patient with pre-existing left bundle-branch-block, complete heart-block ensues.[2] In one such reported case which required implantation of a permanent pacemaker, His-bundle recording confirmed the site of block as distal to the bundle of His, thus providing electrophysiologic documentation of bilateral bundle-branch-block as the cause of complete heart-block.[2] We wish to report another case of complete heart-block occurring transiently during right-heart catheterization in a patient with pre-existing left bundle-branch-block and prolonged P-R interval which were suggestive of bilateral bundle-branch-block,[3] but in whom atrial pacing and His-bundle electrograms during normal A-V conduction failed to induce bilateral bundle-branch-block.

Report of Case

A 61-year-old white man was admitted for cardiac catheterization and coronary arteriography for evaluation of pain in the chest.

The ECG showed left bundle-branch-block and a P-R interval of 0.30 sec (Fig. 1A).

During right-heart catheterization when the catheter traversed the tricuspid valve complete heart-block developed (Fig. 1B). Normal A-V conduction returned after withdrawal of the catheter. A bipolar electrode-catheter was advanced to the right ventricle, and complete heart-block again developed. Demand ventricular pacing was immediately started (Fig. 1C) and continued for 90 minutes until normal A-V conduction returned.

Coronary arteriograms revealed a moderately severe obstruction in the proximal left anterior descending artery.

His-bundle electrogram and atrial pacing were performed percutaneously through the right femoral vein.[4] At a sinus rate of 100/minute the A-H interval was 105 msec and H-V interval 70 msec (Fig. 1D). Following atrial pacing at 160/min, 1:1 A-V conduction persisted, with the A-H interval prolonged to 140 msec, but the H-V interval remained unchanged (Fig. 1E).

Discussion

Right bundle-branch-block is a well-known complication of right-heart catheterization.[1,2] In general, its occurrence is of no serious consequence because it is transient and usually disappears if the catheter is promptly removed from the right ventricular endocardial surface where the right bundle-branch lies. However, if such a patient already had left bundle-branch-block, production of right bundle-branch-block would then lead to bilateral bundle-branch-block with all the serious consequences of suddenly developed complete heart-block.

Subclinical involvement of the right bundle may be present in patients with left bundle-branch-block and may be manifested in the electrocardiogram by a prolonged P-R interval.[3] These patients are prone to develop complete heart block.

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His-bundle recording and atrial pacing in patients with prolonged P-R interval and bundle-branch-block have allowed us to delineate more accurately the sites of conduction delays.[5] Patton and associates,[2] using His-bundle electrography, localized the block as distal to the bundle of His in their patient. Although our patient had complete left bundle-branch-block and a prolonged P-R interval, atrial pacing failed to reveal any functional disturbance of conduction in the right bundle-branch system. It may be postulated that the cause of H-V prolongation in our patient with left bundle-branch-block lies in prolongation of conduction time distal to the His-recording site but proximal to the branching portion of the His-bundle.

Therefore, we conclude that P-R prolongation in left bundle-branch-block does not necessarily indicate additional right-bundle-branch involvement, that subclinical impairment of right bundle-branch conduction needs not be a predisposing factor in the development of complete heart-block in a patient with pre-existing left bundle-branch-block, and that mechanical trauma of a normal right bundle-branch by the catheter during right-heart catheterization is sufficient to precipitate complete heart-block in a patient with pre-existing left bundle-branch-block. The experiences with our patient and that of Patton and associates[2] seem to indicate that a demand ventricular pacemaker should always be on standby in a patient with bundle-branch-block when cardiac catheterization is performed on the side bearing the opposite bundle branch.

Summary

A patient with left bundle-branch-block and prolonged P-R interval developed transient complete heart-block during right-heart catheterization. Although a combination of left bundle-branch-block and prolonged P-R interval usually indicated impaired conduction in the right bundle-branch, atrial pacing and His-bundle recording in our patient failed to induce bilateral bundle-branch-block. A demand ventricular pacemaker should be on standby in a patient with bundle-branch-block when cardiac catheterization is performed on the side bearing the opposite bundle branch, irrespective of the functional integrity of the latter.

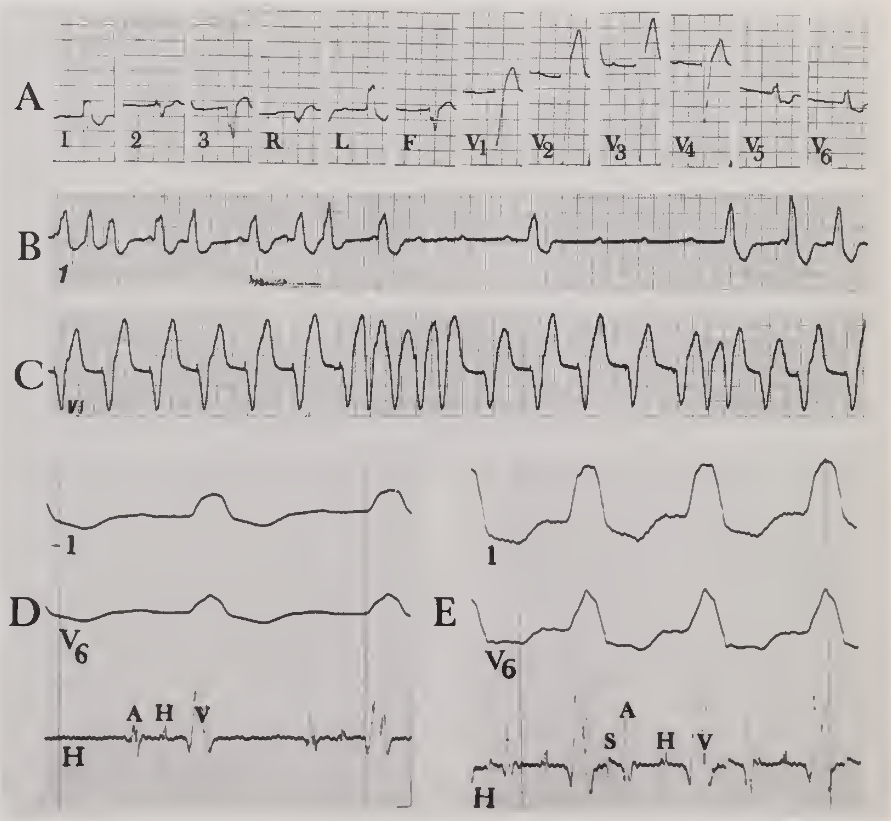

FIG. 1. (A) 12-lead ECG showing left bundle-branch-block and prolonged P-R interval (=0.30 sec.). (B) Lead 1 showing transient complete heart-block during right-heart catheterization. (C) Lead V_1 showing demand ventricular pacing during complete heart-block. (D) Simultaneous leads 1 and V_6 with His-bundle electrogram (H) at sinus rate of 100/min. A = atrial activity. H = His-bundle activity. V = ventricular activity. The time lines are at one-second intervals. (E) Simultaneous leads 1 and V_6 (at double standardization) with His-bundle electrogram (H) during atrial pacing at 160/min. S = stimulus artifact.

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Amenorrhea: Symptom of Endogenous Depression

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Amenorrhea may be the presenting symptom of an underlying intrinsic depressive disorder. Partly because of the association of peculiar concentrations of urinary steroids, affective illness is rarely, if ever, considered in the differential diagnosis of pathological amenorrhea. The following case is presented in an effort to bridge the separation between psychiatry and other medical specialties; especially gynecology and endocrinology. It is hoped that identification of the depressed individual will not only ensure prompt and gratifying recovery but will stimulate new avenues of investigation into etiological relationships of one of the most common and disabling "mental" illnesses.

Report of Case

H. C. was "discovered" by an astute pediatrician. Dr. James Stallings was puzzled by H. C.'s frequent complaints about her three year-old son's health and behavior. He found the child delightfully normal; his diagnosis—"anxious mother." Recognizing parental attitudes to be a significant factor in child development, he and several other pediatricians in the Northern Virginia area developed their own postgraduate educational program in the behavioral sciences. They invited psychiatrists to meet with them one evening each week. During these weekly seminars, group therapy techniques appropriate to pediatric practice were introduced, and selected parents were referred for group counseling. Dr. Stallings suggested that H. C. and her husband join the first group.

Initially H. C. did not participate; she seemed preoccupied. Her mask-like face and staring expression were clues to a severe underlying depression. During the discussion which followed the group session, four pediatricians reported that H. C. made them uncomfortable; all had ignored her, fearing she would burst into tears. The psychiatrist recommended she be referred for immediate evaluation and individual therapy; she appeared too sick to continue in the group.

In private sessions the diagnosis of a severe cyclic depression dating back to childhood was confirmed. H. C. recalled that her childhood was marred by her mother's periodic "depressions" for which there seemed to be no effective treatment. During these depressions H. C. learned to avoid her mother, who was inaccessible emotionally. By her late teens, H. C. experienced feelings of sadness, which came over her without apparent cause. Determined not to follow her mother's example and take her feelings out on others, she developed unusual external control, yet harbored feelings of despair and worthlessness. Following high school graduation she abandoned plans for furthering her education when she developed reading difficulties. She could not concentrate or remember what she had just read. In retrospect she viewed her early marriage as an escape from incapacitating insecurity and depression.

Menarche had occurred at age 12 and appeared normal, but after a year menses became irregular, occurring approximately six or eight times a year. Shortly after her marriage at 19 the menstrual periods ceased, but she was not pregnant. At 21 she was cycled on Enovid for six months with good withdrawal bleeding but no resumption of normal spontaneous bleeding. Several trials of thyroid extract from age 16 to 20 were without success.

At the age of 24, following five years of amenorrhea and infertility of unknown cause, she was referred to the National Institutes of Health. On March 28, 1967 H. C. was admitted to the National Cancer Institute, Endocrinology Branch, in Bethesda. Review of symptoms was unremarkable except for a mild cold intolerance. Physical examination, which included neurological evaluation, revealed an attractive young female with no abnormalities. The weight on admission was 100 pounds, the blood pressure 90/60.

Urinalysis and hemogram were normal. BUN, sugar, cholesterol, alkaline phosphatase, bilirubin, SGOT, T-3, T-4, PBI, chest and skull films were within normal limits; VDRL was nonreactive. Urine for gonadotropin was less than 10 mouse uterine units. Buccal smear revealed 10% sex chromatin body. The only significant endocrinological abnormality was elevation of the 24-hour urinary 17-hydroxysteroid excretion to 19.1, and 17-ketosteroid excretion was 18.1.

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Nonproprietary and trade names of drugs: Imipramine hydrochloride: Tofranil. Geigy Pharmaceuticals (Ciba-Geigy). Norethynodrel with mestranol: Enovid. G. D. Searle and Company.

The hospital course showed a flat basal body temperature; there was good rise in basal temperature in response to progesterone. Endometrial biopsy performed the day of initial progesterone administration was considered unsuccessful because it revealed "no tissue." Very slight spotting, requiring no pads, occurred two or three days after progesterone administration; otherwise no withdrawal bleeding occurred. A Papanicolaou smear was negative, with a karyopyknotic index of 5%. Culdoscopy revealed a proliferative endometrium. Ovarian biopsy showed no response to clomiphene stimulation and no pathologic diagnosis. After 30 days H. C. was discharged to be followed in the outpatient department. The diagnostic impression was "secondary amenorrhea, probably hypothalmic-pituitary etiology. Adrenal hyperfunction as suggested by elevated 17-ketosteroid must be eliminated." H. C. later withdrew from the NIH study because she was "too depressed to go on with all of the tests."

The next two years became increasingly difficult. The depression became worse, and periodically H. C. fought suicidal impulses through prayer. She later expressed her feelings as: "I felt I was losing my mind and could do nothing about it." As her own prophylaxis against "insanity" she devised a program to exercise her reading abilities. She disciplined her mind by forcing herself to read every article on the front page of the newspaper and then wrote what she could remember, checking and rechecking her notes with the original until she grasped the meaning. At the time of her psychiatric referral her daily routine consisted of getting her husband off to work, studying the newspaper an hour or so, and sleeping until time to prepare dinner. Her husband reacted to her increasing apathy and irritability by working later and later until he finally was returning home around 10:00 p.m.

Resigned to permanent sterility, H. C. and her husband adopted an infant boy, but by the time he was 3 H. C. could no longer care for him. The child's lively curiosity was a marked contrast to her overwhelming fatigue, and his demands pushed her to the brink of suicide. She later described her situation: "I was hanging on by my fingernails." From 1:00 a.m. on she lay awake reviewing her inability to cope with her son. As a final desperate measure she consulted Dr. Stallings, the child's pediatrician, hoping he would sedate the youngster; help came just in time.

At the time of referral, psychiatric evaluation revealed a seriously depressed, suicidal young woman. During therapy some interesting facts concerning her family history were revealed. H. C.'s mother flew from Michigan to Washington ostensibly to find out what was happening to her daughter, but covertly seeking help for herself. On examination, the mother was moderately depressed and was referred to a psychiatrist in Ann Arbor under whose care she remains; her medical care included administration of antidepressive drugs. H. C.'s maternal grandmother was also evaluated because of depression, but she refused treatment. H. C. was the youngest of three siblings; the eldest brother died in an auto accident at 18 years of age. The surviving brother, now 34, dropped out of school at the tenth grade because he could not concentrate and was unable to sleep. One of the brother's three children has shown poor academic achievement despite a very good IQ.

On initial examination hospitalization was considered; however, in view of the patient's remarkable strength and the eagerness with which she entered into the psychotherapeutic relationship, it was elected to treat her as an outpatient. In February 1970 conventional doses of imipramine hydrochloride were begun. No improvement in sleep or subjective symptoms was noted after eight weeks of treatment, nor were usual side-effects observed. The pupils remained small, and no dryness of the mouth or postural hypotension was noted. Because of the diagnosis of endogenous depression, imipramine was increased until some change was noted. It was not until a total dose of 400 mg. was reached, four months after the onset of therapy, that subjective symptoms of depression disappeared; by June 1970 H. C. was feeling well and sleeping through the night.

Although no side-effects were noted on 400 mg. of imipramine daily, the dosage was decreased, largely because of PDR's warning that "dosages over 200 mg./day are not recommended." Whenever the dosage was lowered to 300 mg. daily H. C. promptly became depressed although never as severely as prior to therapy. Substitution of other anti-depressive agents proved less effective than imipramine. Whenever the daily dose was increased to 400 mg., subjective depression disappeared, sleep appeared uninterrupted at night, and the patient was awake all day.

Some significant details noted during the course of treatment are summarized: on initial examination only slight breast development was present. Fat distribution appeared to be independent of estrogen influence. After three months of imipramine, significant breast enlargement was noted and the patient discarded her padded bra. During the fifth month of treatment one episode of euphoria was described during which time there was heightened sexual interest and sexual activity. At the beginning of the sixth month of therapy, alteration in dream pattern occurred and the patient reported terrifying dreams; this coincided with an increase in imipramine above 400 mg. daily. Seven months after the onset of therapy acne developed over the face, shoulders, and back; it was severe enough to be treated by a dermatologist with tetracycline. The patient had had no acne during her adolscent years. With treatment it was apparent that remarkable feminization had occurred; it is equally significant that at no time, either prior or after treatment, had the patient shown any signs of virilization.

Although no longer depressed on 400 mg of imipramine daily, it was not known whether optimum drug response had been achieved. Over a period of a few weeks the maximum tolerated dose of 800 mg. imipramine daily was achieved; dryness of the mouth was severe and dizziness precluded further increase. Since no further improvement was noted at doses between 400-800 mg. daily, a 400 mg. "maintenance" was adopted. In October 1970, nine months after the onset of therapy, H. C. experienced her first menstrual period in nine years!

After 10 months on imipramine considerable thigh enlargement developed and the patient complained about weight fluctuations which appeared somewhat independent of diet. In January 1970, prior to treatment, weight was 105 pounds; by April, a five pound weight gain was noted. In July weight was 118 pounds. In October, at the time of the first menstrual period, weight was 124 pounds. From October 1970 to January 1971 weight fluctuations between 118 to 135 pounds were noted. Over the next year the weight stabilized at 132 pounds.

Although the patient was delighted with her recovery, her physician had been growing increasingly anxious. Consider FDA's reaction to a child psychiatrist's treating an adult with enormous amounts of imipramine! Dr. William E. Bunney, Chief, Section on Psychiatry, Laboratory of Clinical Science, NIH, was consulted, and he kindly interviewed H. C. and offered helpful suggestions and support. Dr. Bunney believed H. C. suffered from an underlying manic-depressive illness and that the addition of lithium carbonate might prevent a hypomanic state and prevent a recurrence of the depression. Thanks to Dr. Bunney's encouragement and to H. C.'s trust, lithium carbonate was started and ultimately was successful. Initially 600 mg. of lithium carbonate, t.i.d., were required to keep blood levels within therapeutic range. After stabilization on lithium, imipramine was tapered, and after six months of combined therapy imipramine was discontinued. H. C. remained well on a maintenance of 300 mg. lithium carbonate in the morning, 300 mg. at 4:00 p.m. and 600 mg. at bedtime. Urinary steroid excretion returned to normal; a comparison with pre-treatment (NIH) levels is shown in table 1. Subjective improvement coincided with objective improvement. Social isolation gave way to involvement in community activities. Short-term memory returned to normal, and reading was rapid. The patient enrolled in a business school and completed an entire secretarial course in less than half the normal time. Although active, she did not appear to be abnormally energetic. As irritability and fatigue disappeared, the therapist was surprised to discover a delightful sense of humor and keen intellect which had been masked entirely by the illness.

Menstrual periods occurred regularly every 28 days; however, final proof of gynecological cure came eight months after the institution of lithium carbonate. The patient's self-diagnosed "lithium toxicity," nausea, occasional vomiting, and a missed menstrual period turned out to be a normal 10-week intrauterine pregnancy.

Discussion

Symptoms. H. C.'s history brings into focus many common symptoms of depression which are frequently overlooked or are not considered to be an important feature of the depressive syndrome. Early symptoms which I believe are almost universal, include the following:

1. Sleep: early morning awakening has been considered one of the cardinal signs of depression; however, hypersomnia is seen perhaps as frequently. At the height of H. C.'s depression she was sleeping on and off most of the day; hypersomnia was present despite the presence of early morning awakening. In H. C. and other seriously depressed patients I have treated, there appears to be an almost day-night reversal of the normal sleep process, suggesting a disturbance in circadian rhythm.

2. Memory: reading difficulties of a characteristic nature are present and may be the presenting complaint in children. The patient states he can read the words but must turn back a page or paragraph because he can not remember what he has just read. On

TABLE I: 24-Hour Steroid Excretion

	17-ketosteroids (normal: 5-15 mg./24 hrs)	17-hydroxycorticosteroids (normal: 4-16 mg./24 hrs)
1967 (pre-treatment)	18.1 mg./24 hr	19.1 mg./24 hr
1971 (post-treatment)	14.4 mg./24 hr	12.2 mg./24 hr

psychological testing this symptom can be documented by the observation of short-term memory dificit on I.Q. testing.

3. Weight: mild to moderate obesity appears to fluctuate out of proportion to changes in caloric intake. Although the relationship between diet and weight is difficult to impossible to document reliably in an outpatient setting, there appears to be sufficient information from the patients' relatives to warrant investigation into neuro-endocrinological mechanisms in weight control. In my experience weight-loss appears to be prominent only in individuals suffering depressions of a severe nature.

4. Temperature regulation: a mild intolerance to cold is often overlooked in routine histories. This symptom was documented in H. C.'s NIH record. Its significance, in the presence of normal thyroid function, was not understood. With treatment, this complaint disappears.

5. Menstrual irregularities: in my experience in both pediatric and psychiatric practice, gross menstrual irregularities in an otherwise healthy woman are almost pathognomonic for depression. Any physician treating adolescents should be aware of this association. Many unnecessay X-rays, pelvic examinations, and endocrinological studies might be avoided by a careful history alone.

Treatment. I consider treatment of depression somewhat analogous to the treatment of diabetes; unfortunately, urine and blood tests are not yet available as guides to drug therapy. Successful treatment appears dependent upon careful interrogation of the patient and the physician's observations. Physical changes are most conspicuous in the female and are of such a nature as to suggest that altered metabolism interferes with estrogenic function. Prior to therapy there is little breast tissue. With treatment, the breasts become full, and typical estrogenic fat distribution occurs. As in the diabetic, drug dosage appears to change with stress; emotional stress is to the depressed as a Roman feast is to the diabetic. Sensitivity to the patient's complaints and warning signs permit appropriate medication adjustments; the physician who listens attentively can contribute much to the understanding of early detection and prevention of disability. Contributions of medical specialties other than psychiatry should provide further insight into the basic neuropathology and etiology of this common and disabling illness.

Classical or analytically orientated psychotherapy appears contraindicated in severe depressive illness. Psychotherapy promotes introspection; the depressed individual is already too introspective. Supportive psychotherapy appears to be exceptionally rewarding.

Finally, a word concerning medicolegal implications in the treatment of depression: There seems to be no more rationale to the limitation of dosage schedules as recommended by PDR than there would be to the limitation of insulin doses. Such cautions preclude optimal patient care. Dosage restrictions serve to denigrate the clinician. Regulatory surveillance by FDA stifles sound clinical therapeutics and leaves, instead, an undertreated patient who has been denied the benefits so easily afforded him. Imipramine is a safe drug. Its reversible side-effects—postural hypotension, dry mouth, constipation and impairment of (visual) accommodation are annoying enough to the patient to serve as built-in signals of overdose. Articles concerning hazards of overdose are rare and misleading in the context of the millions of persons receiving the drug for periods of years. The recent premature reporting of imipramine-induced phycomaelia in three offspring in Australia seemed to reflect journalistic hysteria and a search for the sensational rather than sound medical reporting.

Depressed patients are metabolically different. As seen in the case history of H. C., severe endogenous depression can be a cause of infertility. Any report of birth defects caused by imipramine must take into account the fact that the drug is used for an illness which in itself affects normal reproduction. If and how a partially treated or a mild endogenous depression affects the fetus, is a subject for speculation and research.

No federal medical surveillance has yet been able to substitute for clinical competence. The case presented serves to point out how federal control on medical practice can stifle clinical competence and prolong the suffering of the patient.

Summary

The case of a young woman in whom amenorrhea was the presenting complaint, emphasizes the protean nature of endogenous depression and suggests that specialities other than psychiatry might have much to offer concerning the pathogenesis and treatment of this common and disabling "mental" illness. Remarks concerning irrational restriction of imipramine dosages have been included.

Diagnostic Ultrasound in Cardiology[*,†]

RONALD H. COOPER, M.D. AND GEORGE R. LEOPOLD, M.D.

Diagnostic ultrasound is a rapidly developing, non-invasive technique with important applications in clinical cardiology. It employs inaudible, high-frequency (greater than 20,000 Hertz) vibrations produced by activation of a piezo-electrode crystal and directed through tissues being examined. At interfaces between media of differing acoustical impedance (such as between blood and tissue) a small portion of the mechanical energy is reflected, detected by the same crystal acting as a receiver, and converted to electrical impulses suitable for oscilloscopic display. The time elapsed between transmission and reception of the ultrasound pulse reflects the distance of the interface from the transducer. Returning echoes are displayed conventionally as an ultrasound cardiogram (UCG) with distance on the vertical axis and time on the horizontal. Time-distance markers and a simultaneous electrocardiogram are incorporated for reference. Patients are studied in the supine or partial left-lateral decubitus position with the transducer positioned in the third to fifth intercostal space, one to six cm from the left sternal border. The UCG provides diagnostic information useful in assessing pericardial effusions, valve motion, chamber dimensions, and ventricular performance.

Pericardial Effusion

The easiest interface to identify on the UCG is that between the posterior heart wall and the surrounding lung. In the normal heart epicardium and pericardium are so closely adherent that they are represented by a single echo. However, when fluid accumulates in the pericardial space, these surfaces are separated, and abnormal, additional interfaces are created. Diagnostic ultrasound recordings reveal echoes from both the non-pulsatile pericardium and the pulsatile epicardium. Between them is an echo-free space which represents the periocardial effusion. In many series the UCG has proven sensitive in the detection of small to moderate pericardial effusions, and in several centers ultrasound recording has become the diagnostic procedure of choice when this condition is suspected.

Valve Motion

Echoes from the anterior leaflet of the mitral valve produce a characteristic normal pattern. Movement of the leaflet into the left ventricular cavity is seen on the UCG as anterior motion, while movement toward the closed position appears as posterior movement. During the rapid filling phase of early diastole the anterior leaflet of the mitral valve opens quickly into the left ventricle. As the lower chamber fills, this leaflet floats toward a closed position, only to be opened widely again as contraction of the left atrium proceeds. At the onset of ventricular systole the mitral valve is slammed shut and remains closed throughout the ejection period. On the UCG the diastolic movement of the leaflet resembles an "M" with the first peak somewhat higher than the second. The systolic position of the leaflet appears as a straight line sloping gradually upward and joining the bases of two diastolic "M's".

Stenosis of the mitral valve is associated with a decrease in the velocity with which the anterior leaflet floats toward a closed position in mid-diastole. Significant hemodynamic impairment is associated with velocities of less than 35 mm/sec. The total amplitude of excursion of the leaflet from the closed to the fully open position is a useful indicator of the mobility of the valve. Patients found to have immobile leaflets at surgery usually exhibit valve-leaflet amplitudes of less than 15 mm. Left atrial enlargement, a concomitant of several atrial valve diseases, can be assessed by recordings made with the ultrasound beam directed through the aortic root to the posterior atrial wall and measurements of the distance between the two corresponding echoes. Mitral-valve prostheses in patients treated surgically can be evaluated readily by ultrasound. The dimensions of the poppet, the extent of ball excursion, and the velocity with which the ball moves can be quantified. Accordingly, the UCG facilitates detection of changes suggesting ball-variance or the presence of thrombosis.

Patients with the syndrome of mid-systolic click and late systolic murmur

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exhibit prolapse of either the posterior or both leaflets of the mitral valve. On the UCG this prolapse appears as an arrest of the gradual upward slope of the valve leaflet normally seen during systole and posterior systolic movement of one or both leaflets, usually in a smooth, semicircular pattern. A different disturbance of the normal systolic movement of the anterior leaflet is exhibited by patients with hypertrophic obstructive cardiomyopathy. In this condition contraction of the hypertrophied intraventricular septum appears to pull the anterior leaflet forward into the outflow tract of the left ventricle, thereby creating both an obstruction to outflow and mitral-valve incompetence. This forward movement of the leaflet is visualized on the UCG as a systolic, anteriorly directed, semicircular bulge in the anterior mitral-valve-leaflet tracing.

The UCG has proven useful in the diagnosis of atrial myxoma. Normally the space immediately posterior to the anterior valve is echo-free. If, however, a pedunculated atrial tumor falls into the mitral-valve orifice during diastole, the clear area is obliterated by characteristic echoes and the diastolic movement of the leaflet is impaired, producing a pattern resembling that seen with mitral stenosis.

Additional Applications

In the normal heart the posterior wall of the left ventricle and the ventricular septum approach each other during systole and move apart in diastole. Empirical formulae based on end-diastolic and end-systolic dimensions can be used to estimate ventricular volumes and ejection fractions. The velocities at which the septum and posterior wall approach each other during systole can be determined and utilized as an index of myocardial contractility. Such an index, obtained non-invasively, is applicable to serial assessment of a critically ill patient's cardiac status following an acute insult such as myocardial infarction and its response to therapy.

Several specific congenital cardiac anomalies can now be identified by characteristic UCG patterns. Thus, the small right-ventricular cavity and exaggerated diastolic, tricuspid valve movement in Ebstein's anomaly and the absence of an intraventricular septum in patients with a single ventricle are identifiable and helpful.

Although satisfactory echocardiograms cannot be obtained from all the patients, especially those who are obese or those with marked chest-wall or emphysematous lung changes, ultrasound has become a valuable diagnostic tool. The technique is non-invasive and a-traumatic. It can be utilized serially as frequently as desired, and it provides permanent records suitable for quantitative diagnostic analysis pertinent to a variety of cardiac disorders.

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Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders, athetosis, stiff-man syndrome, convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence. In pregnancy, lactation or women of childbearing age, weigh potential benefit against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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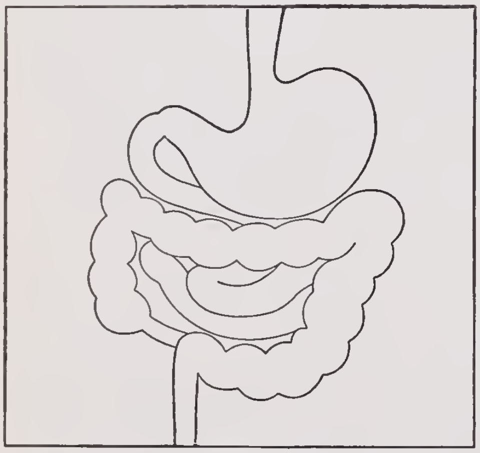
To help you manage excessive psychic tension

in “Gasspastic” conditions



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can do more

Contraindications: Anticholinergics should not be used in patients with glaucoma, known prostatic hypertrophy, or pyloric obstruction. Urinary retention may indicate the presence of prostatic hypertrophy. If it occurs, the dose should be reduced or the drug withdrawn. Also contraindicated in patients with known hypersensitivity to one of the components.
Side Effects: Dryness of the mouth, blurred vision, dysuria, skin rash, constipation or drowsiness may occur.

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Failure to Disclose Risk as Malpractice

Many people who have undergone surgery or other serious medical treatment can recall their ignorance of the technical nature of the operation and the particular risks that threatened disability or death. This is due particularly to the failure or refusal by many physicians to disclose more than the most basic information to their patients before treatment. Such physicians should be keenly aware that, if injury results from treatment entailing a high degree of risk, an increasing number of courts will award damages to the patient on the ground that the physician wrongfully withheld information from the patient about his case.

In a 1972 District of Columbia case, a patient sued his doctor on the grounds of alleged negligence in the performance of a laminectomy and failure to inform him beforehand of the risk of paralysis involved. The lower half of the patient's body was paralyzed as a result of the operation. The doctor testified that, in his opinion, the patient's disability stemmed from his preoperative condition; the doctor admitted that trauma could be a cause of paralysis. The doctor further testified that, even in the absence of trauma, paralysis could be anticipated somewhere in the nature of one percent of the laminectomies performed, a risk he termed "a very slight possibility." The doctor believed that communication to the patient of that risk would not have been good medical practice because it might have deterred the patient from undergoing needed surgery and might have produced adverse psychological reactions which could preclude the success of the operation. Despite the doctor's explanation, the U.S. Court of Appeals held that the failure to reveal the risk resulted in a clear case of violation of the physician's duty to disclose.

The theory of liability for breach of the duty to disclose medical information—sometimes called the doctrine of informed consent—appeared in a 1960 Virginia case, in which a patient recovered $6,000 from a surgeon who, although suspecting that a sponge had been left in an operative wound, failed to reveal his suspicion to the patient or to another doctor, and did not suggest or order an X-ray. In fact, the origin of the doctrine of informed consent can be traced to the dictum (that is, an aside not necessary to the decision in the case) of a 1918 Virginia case, in which the court, finding negligence in the administration of X-ray treatment, analyzed the issue in terms of the doctor's failure to warn of the risks involved in the then new technique.

The legal wrong (or "tort") for failure to disclose medical information is independent of any improper treatment and usually takes place before the physician treats the patient. The tort arises not from the physician's negligent performance but from the procedure selected. The injury is simply a bad result which can be foreseen but cannot be avoided by anyone in the profession. Although a physician should not be (and generally has not been) held liable solely because he was unsuccessful, recovery is allowed for unexpected bad results where the physician could have warned the patient of the risks prior to treatment.

Underlying the doctrine is the theory that the right of a person to protect or disregard his own health is inherent in the basic rights of bodily freedom and individual choice. Although the health of the individual citizen is one of society's greatest concerns, it is generally recognized that a competent adult cannot be compelled by the state to submit to medical treatment against his will. The relationship between a physician and a patient is a consensual one wherein the patient knowingly seeks the assistance of the physician and the physician knowingly accepts him as a patient. A physician therefore cannot treat a patient unless he has first obtained the patient's consent to act. Treatment without consent constitutes a "harmful touching"—that is, a battery. This rule thus protects the patient's right to choose what should be done with his body; that the treatment may be beneficial to the patient is not of primary significance.

The physician has a fiduciary obligation to his patient. He is expected to place the interests of his patient above his own. The professional ethic demands that the physician deal frankly and honestly with his patient in obtaining the patient's consent.

Consent may be obtained in several ways. The physician may obtain the *express* consent of the patient either orally or in writing. Consent may be *implied in fact*, as where a patient rolls up his sleeve for an injection, or it may be *implied in law*, as in the case of an emergency where the patient is physically or mentally unable to give his express consent. Consent may also be given by a parent or guardian for a child or an incompetent.

Despite the relatively simple recitation of these rules, the conclusion should be avoided that the establishment of a valid consent is merely mechanical. The right of a patient to decide what to do with his body necessarily implies the right to receive and comprehend the information necessary to make an intelligent choice. Thus, a consent is valid only to the extent that it is intelligent or informed, with a clear understanding of what is to be done and of the risks involved.

Unfortunately, no precise formula exists as to the scope of the physician's duty to inform his patient of the circumstances surrounding contemplated medical procedure. Most courts hold that a physician must disclose what a reasonable medical practitioner of the same school and same or similar community would have disclosed. Other courts do not accept the defense of the community standard and have adopted a rule requiring that disclosure which a reasonable medical practitioner would have made under the same or similar circumstances.

The case law reveals various factors which will materially affect the extent of the duty. Primary among these factors is the likelihood and predictability of the result which occurred. Thus, where the consequences of risk were inevitable or inherent, courts find a clear duty to warn. In those cases involving more remote consequences, the rule seems to be that as long as the physician warns the patient that any surgical procedure involves some risk of unfortunate consequences, he has adequately fulfilled his duty; yet, in the District of Columbia, as evidenced above, such generalized warning would not suffice to insulate the physician from potential liability.

Other factors affecting the extent of the physician's duty to disclose include the feasibility of alternative methods, the interest shown by the patient, and the existence of an emergency.

The disclosure of every conceivable risk is not required. Since the average patient is presumably aware of the dangers inherent in any treatment, the duty is essentially reduced to a disclosure of the dangers peculiar to the treatment and of which the patient may be unaware.

A therapeutic privilege in the physician to withhold full and frank disclosure of possible harmful effects has generally been recognized where such disclosure would tend to jeopardize the recovery of an unstable, temperamental, depressed or unduly apprehensive patient. But the burden will be on the defendant-physician to show that limited or non-existent disclosure was appropriate under the circumstances. Therefore, the physician should, if possible in such situations, make full disclosure to close relatives of the patient. Disclosure cannot be withheld or limited merely because the patient might, on learning of the risks involved, decline treatment, because such right to decline is the specific fundamental right protected by the informed consent law.

Conclusion

In over 20 jurisdictions, including D.C. and Virginia, standards have evolved for the cause of action for failure of a physician to secure his patient's informed consent to treatment. The action is based on the patient's allegation that if the doctor had disclosed the risk of resulting injury, the patient would not have consented to the procedure. Liability is found where the physician failed to make a reasonable disclosure of all significant facts under the circumstances.

A failure to disclose will not necessarily suggest a neglect of duty in all instances. The welfare of the patient is the paramount consideration. The informed consent rule emphasizes the duty to forewarn of the possible and probable consequences of new, experimental or unusual techniques and of possible serious complications of standard methods of treatment. The physician is allowed a certain amount of discretion as long as its exercise is consistent with the full and frank disclosure of facts necessary to an informed consent.

RONALD H. KISNER

Edited by Stefan F. Tucker of Arent, Fox, Kintner, Plotkin and Kahn, Washington, D.C.

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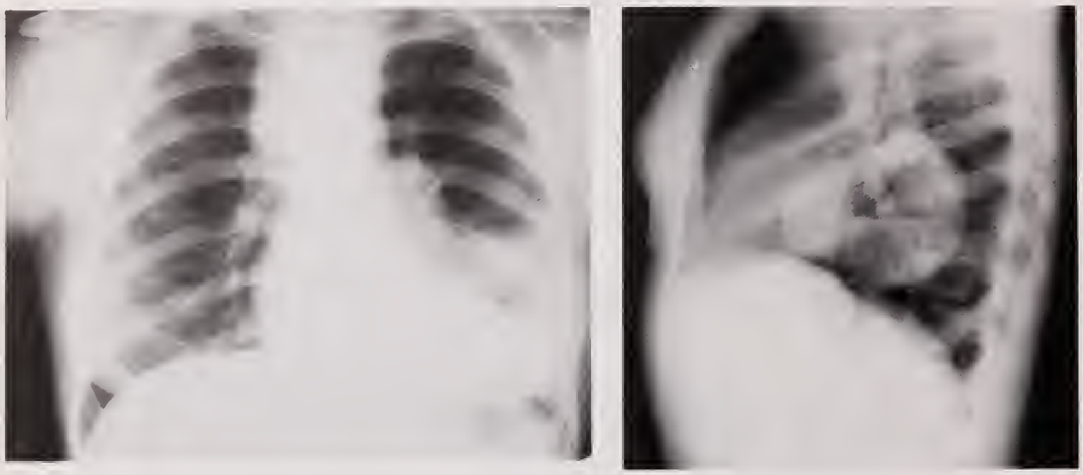
THE VIEW BOX

Prepared by
The Department of Radiology
Washington Hospital Center

A 19 year old white female presents with a history of productive cough of several weeks duration associated with fever, weight loss and general malaise.



(For diagnosis see page 768)

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d.c. Health NOTES

The Public Health Laboratory Solves Health Mysteries

Three hundred people in the United States became ill with symptoms of abdominal pain, nausea, and vomiting. One third of these people lived in the District of Columbia. The incident took place several months ago. The culprit—Entero pathogenic Escherichia coli 0124: B17—was first identified in the Community Health and Hospitals Administration (CHHA) laboratory. The organism was isolated in the stool specimens of those who became sick and was later traced to cheese which was exported to the United States.

The laboratory workers, specialists in chemistry, pathology, microbiology, and serology, perform their duties without fanfare or public attention. Through the year, they complete more than one-half a million tests, searching, analyzing, and recording data that help solve public health problems.

In addition to testing procedures, laboratory personnel experiment with new techniques to find ways of reducing cost. Recently, a solution was prepared which will reduce by four-fifths the cost of 30,000 annual Sickle Cell Anemia screening tests conducted by the CHHA. Commercially prepared solutions for this test now cost approximately fifty cents each. Using the new technique, each test will be reduced to approximately ten cents.

Another procedure developed in our laboratory will save money as well as make it less difficult to obtain a blood sample for lead poisoning. Instead of a veni-puncture, which is extremely difficult with youngsters who are one, two, three, or four years old, a finger-stick method can be utilized. No one else in the country is using this technique as far as we can determine, and we have demonstrated to our satisfaction that sufficient blood can be drawn to adequately perform the screening test for this health problem. The ease with which this test is administered is a break-through in finding those youngsters who are lead intoxicated.

The CHHA Bureau of Laboratories provides services for the Department of Human Resources, other D.C. government agencies, public and private hospitals, private physicians, and other persons involved in the control of disease. In addition to the central laboratory located in the Municipal Center, other laboratories provide services in the Neighborhood Health Centers, D.C. General Hospital, and Glenn Dale Hospital, all part of the CHHA.

Laboratory specialists in microbiology examine human and animal specimens and food and milk samples for bacteria, viruses, fungi and parasites. Personnel assist in insuring that water and milk supplies are in compliance with U.S.P.H.S. standards. Tests are made for fecal coliforms as an indicator of sewage contamination. Milk is tested for compliance with general sanitary quality and for the presence of antibiotics and other antibacterial substances. Food suspected of involvement in gastrointestinal upsets and other foodborne episodes are analyzed to identify the organism.

Drug susceptibility and virulence investigation are important functions of the microbiologists. Also, tubercle bacilli and atypical Mycobacteria viruses including Enteroviruses, Respiroviruses and Adenoviruses are isolated and identified.

Chemists play an important role in the activities of the Bureau of Laboratories. They identify body samples for evidence of lead, arsenic, mercury, and other toxic metals, in addition to identifying alcohol and drug levels of persons involved in vehicular accidents. Performing chemical profiles, staff analyze body samples for glucose, urea nitrogen, uric acid, triglyceride and cholesterol. Serological tests for syphilis and other diseases are conducted daily on specimens submitted by physicians, clinics, and hospitals. Fluorescent antibody tests also are conducted for febrile diseases as well as for viral and rickettsial diseases. Personnel also provide hematology and immunohematology services. These include blood counts, hemoglobin, blood groupings and pH factoring.

Specimens in exfoliative cytology and histotechnology are examined by specialists in pathology. Cytology specimens are accepted for examination by the Pap smear method. Also processed are samples of the self irrigation technic and Gravlee jet washings. The latter is a newly developed and approved technique.

Cyto-technicians screen cases for review by the pathologist.

The central laboratory serves as the state laboratory in the District of Columbia. As such, personnel are detailed to conduct annual inspections of all independent and private laboratories and tissue banks in the District. Certificates of accreditation are issued to laboratories which permit the continuation of operations.

In addition to its technical functions, the Bureau of Laboratories provides opportunities for career development for persons not previously exposed to laboratory skills. Through the Work Incentive Program, women receiving welfare assistance have an opportunity to learn on the job and with sufficient training and experience rise through the ranks to become technicians and skilled technologists.

Physicians interested in touring the facilities of the Bureau of the Laboratories or have questions concerning the Bureau may contact Alston Shields, Ph.D., Chief, Bureau of Laboratories, CHHA at 629-3623.

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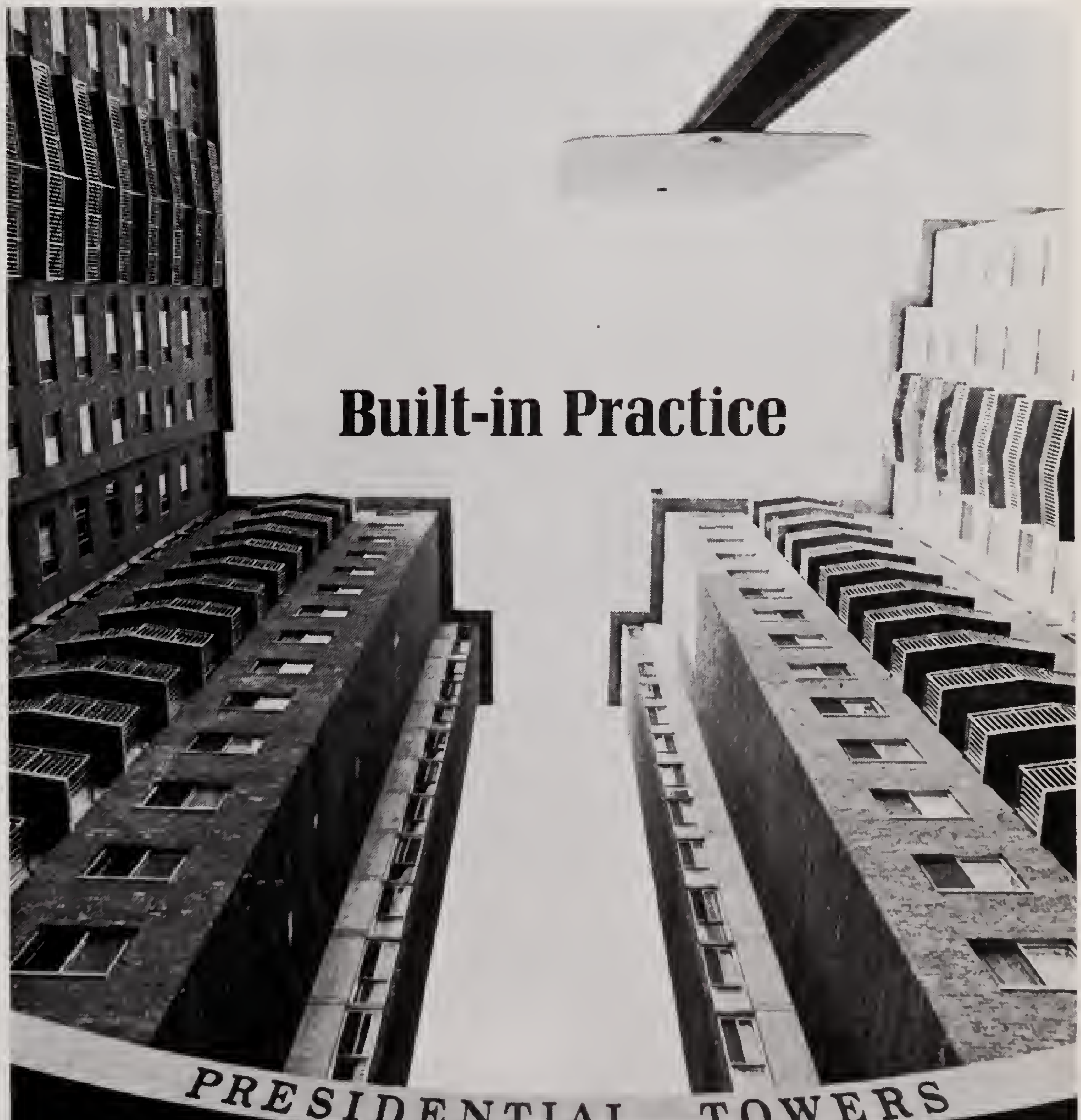

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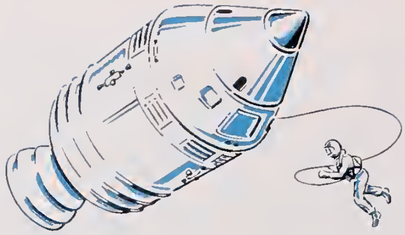

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Transfers

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Associate to Special: Frank E. Gibson, M.D.

Resident-Intern to Active: Bernardo Kotelanski, M.D.

Reinstatements

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book REVIEWS

Hydrocephalus and the Cerebrospinal Fluid. THOMAS HERRICK MILHORAT. Price, $18.50. Pp. 237 with illustrations. Baltimore: Williams and Wilkins, 1972.

The past twenty years have seen a significant extension of our knowledge about the origin and fate of cerebrospinal fluid. These data have mainly been the product of experimentation with improved animal models and the development of radioisotope cisternography. During the same period the successful surgical treatment of hydrocephalus in man has been greatly enhanced as a result of the introduction of unidirectional valves for the shunting of cerebrospinal fluid from the lateral ventricles of the brain to extracranial sites, the cardiac atrium in particular. Up-to-date information on both subjects has been published widely in journals and monographs.

This volume, by the Chief of Neurosurgery, Children's Hospital National Medical Center, is unique in that it successfully correlates the anatomical and pathophysiological alterations seen in the brain of the hydrocephalic victim with the accompanying changes in cerebrospinal fluid dynamics. As a consequence, the reader is treated to a unified account of both subjects documented by personal experience and by lengthy bibliographical annotations. After a superb initial chapter on cerebrospinal fluid physiology, five subsequent sections deal in an orderly manner with the pathogenesis, pathology, pathophysiology, diagnosis and treatment of hydrocephalus. The author avoids polemics by presenting the varied attitudes toward controversial subjects while at the same time stating his particular stance on the matter in question.

The illustrative material is carefully chosen to illuminate the text and is of superior quality.

Minor irritants to this reviewer include the insertion of too many footnotes (they appear on 38 pages), the liberal use of the editorial "We" and the failure to include an author index. Nevertheless, this should in no way detract from the fact that this book is must reading for those clinicians involved in the care of hydrocephalics be they pediatricians, neurologists, neurosurgeons, radiologists or physiatrists.

NORMAN H. HORWITZ, M.D.

Symposium on Strabismus. Transactions of the New Orleans Academy of Ophthalmology. HERMANN M. BURIAN, M.D., ET AL. Price, $34.50. Pp. 426 with illustrations. St. Louis: C. V. Mosby Company, 1971.

The 1970 New Orleans Academy of Ophthalmology devoted itself for the third time to strabismus and aimed, as Dr. Hermann Burian stated, "at the practicing ophthalmologist and his practical dealings." The aim has held true. This symposium on strabismus makes a strong contribution toward the understanding of this often complex subject.

Seven distinguished American authorities have contributed twenty well written readable chapters which run the strabismological gamut from the examination of the patient to the physiology of the normal and abnormal fixations to the various horizontal and vertical abnormalities of the specifics of treatment.

Mary Fletcher writes of the natural history of strabismus. Her findings that most intermittent exotropia in unoperated children tends to improve are provocative though they leave unanswered the question of what the words "most" and "improved" means and what the actual statistics are. In view of the general feeling to the contrary, her conclusions lay the ground work for more work on the natural history of strabismus.

Dr. Jampolsky's article on diagnosis with its emphasis on fixation and accomodative control was excellent although there may be those who differ strenuously with his labeling as unnecessary a stereopsis evaluation.

Dr. Parks presented an excellent review of the sensory aspects of strabismus including a fine chapter explaining the monofixation syndrome. Particularly interesting was his challenge to the classic theory of anomalous retinal correspondence. Rather than being confined to the retinal areas upon which project the object of regard (the macula area of the fixating eye and the extra foveal area of the deviating eye), he presents evidence to show that ARC is the cortical adjustment that takes place to eliminate peripheral diplopia and confusion.

Doctors Kanpp and Dunlop presented many worthwhile suggestions in the management of typical, atypical, and seemingly refractory squint problems. The danger of overcorrection in patients with lateral incomitance was stressed. Dr. Burian had good chapters on the management of vergence anomalies and amblyopia.

While in a book of this type the author's bias on his subject is stressed, nevertheless, the wide scope of the material, the fine organization by the editors and the general excellence of the individual chapters make this book a must for the practitioner who is seriously interested in strabismus.

THOMAS FREY, M.D.

Histopathologic Spectrum of Regional Enteritis and Ulcerative Colitis. N. KARLE MOTTET, M.D. Price, $16.00. Pp. 249, with illustrations. Philadelphia: W.B. Saunders Company, 1971.

The book consists of 249 pages of text presented in 10 readable chapters that are profusely illustrated. Of the 160 illustrations, there are 50 gross photographs, 86 photomicrographs, 15 electron micrographs, 5 line graphs, and 4 clinical x-rays. Five of the photographs are in color. The gross illustrations are especially well taken, and the photomicrographs are often at low maginification so that structural alteration is easily evident to the clinical reader. Several tables are included. The references are listed at the end of each chapter; most are recent and the total number of 446.

The text is comprehensive and includes, for both entities, historical aspects, etiologic considerations, clinical features, morphologic examination procedures, gross and histologic characteristics, extra-intestinal lesions, and pathologic consequences including neoplastic sequelae. While attention is given to clinical management, pathologic process is the central theme. In the characterization of regional enteritis, emphasis is given to the segmental distribution of the lesion, transmural involvement of the gut wall, granulomatous nature of the inflammation, cicitrization as the gradual outcome, and frequent extension of the process to mesenteric lymph nodes. The condition occurs on both sides of the ileo-cecal valve, and the author advises that in the colon it should be termed "regional enteritis involving colon" rather than "granulomatous colitis" which fails to distinguish it from several other granulomatous diseases of the large bowel. In contrast, ulcerative colitis is predominantly mucosal, exudative in nature, noncicatrizing, and affects large portions of the colon without frequent extension to mesenteric lymph nodes. The author registers the high occurrence of carcinoma in ulcerative colitis, and cites 21 cases of intestinal cancer in patients with regional enteritis. While he does not declare whether this is coincidence or consequence, the latter appears favored because most of the tumors occurred in the small intestine, most developed at an earlier age than usual, and more than half had regional enteritis for more than a decade before the onset of the cancer.

The book is considerably more than a "histologic spectrum"; it is a comprehensive review of the clinical and morphologic aspects of both conditions as well as those conditions from which both must be distinguished. It is recommended as an extraordinarily fine account of these two puzzling and important intestinal inflammations.

J. CHANDLER SMITH, M.D.

THE VIEW BOX

(from page 756)

DIAGNOSIS: Hodgkins Lymphoma

Note the mediastinal lymph node enlargement and mass lesion in the left lower lobe with a thick walled cavity.

In about 50% of patients there is, usually, bilateral asymmetrical node enlargement. Often, as in this case, the involvement is in the mid-mediastinum but nodal enlargement in the anterior and posterior mediastinum is not uncommon. Pleuro-pulmonary involvement occurs in less than 30% (Fraser and Pare') either as direct extension or in the form of a mass in the lung parenchyma. Cavitation of these masses can occur.

medical annals of the district of columbia

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Living With a Man Who is Dying. JOCELYN EVANS. Price, $4.95. Pp. 143. New York: Taplinger Publishing Company, 1971.

This is the brief personal memoir of a woman whose husband is dying of widespread malignancy. It was never clear whether the primary carcinoma was pancreatic or lung, but this is irrelevant. What is important is the message that many failures of communication between the medical professionals who cared for him and the family occurred. The patient, who was never told the diagnosis but definitely seemed aware of it, and his wife decided to avoid returning to the hospital after an exploratory laparotomy, at which time he was merely opened and closed.

His doctors would have preferred he die quietly and heavily sedated in a hospital back room. But his wife felt compelled to help him live his last days at home. When this course was elected the attending physician and surgeon seemed to avoid the necessary support this required. It is a sad commentary on the doctor-patient relationship when an answering service becomes a protection for the doctor rather than an adjunct to prompt and compassionate care. While these events occurred in England under the National Health Service, I am certain that the same drama is played out in this country and many others.

The love and devotion of this wife were more important to the patient than all the drugs or doctors the world could offer. Many do elect the course she chose, as I learned as a visiting physician for terminal patients at the D.C. Cancer Society. It is not for us to decide how a particular family should handle such a situation, but rather we should provide maximum psychological and medical support no matter which course they choose.

NORMAN K. BOHRER, M.D.

Medical Emergency Manual. MARY E. MOORE, M.D., Editor. Price, $8.95. Pp. 160. Baltimore: The Williams and Wilkins Company, 1972.

This book, as its title suggests, is a handbook of guidelines for the management of the more common major medical problems seen in emergency room practice. It is in no way meant to be all-inclusive, but in dealing with the topics outlined, the text is fairly complete.

There are two major subdivisions of the book. The first deals with the diagnostic approach to the basic problems of coma, shock, chest pain, drug intoxication, and so forth. Each chapter is broken down into history, physical examination and laboratory findings helpful in making a particular diagnosis. The second subdivision deals with the treatment of specific problems of cardiac arrest, arrhythmias, respiratory failure, seizures, metabolic emergencies, and others.

There are no outstanding features of this overview of emergency room problems. The saving grace, however, is that each chapter has a list of references, many of which are from recently published literature. This text may not be suited for the physician already in practice, but it would be a good reference book for medical students, interns and first year medical residents who must learn a form of standard emergency room practice.

MARTIN I. COHEN, M.D.

Practical Electrocardiography, 5th edition. HENRY J. L. MARRIOTT, M.D. Price, $10.50. Pp. 325. Baltimore: Williams and Wilkins, 1972.

Recent advances in electrocardiography necessitate a periodic review of this discipline for even the most seasoned clinician. This Fifth Edition of Dr. Marriott's widely known work is both practical and timely. New sections have been added on recent concepts of the hemiblocks, Wolff-Parkinson-White syndrome, accelerated idioventricular rhythms, His bundle electrocardiography, and artificial pacemakers. The arrhythmias are discussed in a thorough, yet practical manner with emphasis on the monitor lead system currently used in coronary care units.

The text is clear and concise with illustrations arranged to be directly at hand. An occasional aphorism has been added to emphasize key points. References and review tracings are provided conveniently at the end of each chapter.

This book will be of value to the neophyte as well as to the more experienced reader who may wish to update his current knowledge in this field.

LAWRENCE KLEIN, M.D.

Identification of Enterobacteriaceae, 3rd edition. P. R. EDWARDS AND W. H. EWING. Price, $12.00. Pp. 362. Minneapolis, Minnesota: Burgess Publishing Company, 1972.

This soft covered spiral book is an up to date edition of a laboratory manual of the Enterobacteriaceae. Dr. Ewing, who carried on the work of Dr. P. R. Edwards after his death in 1966, has classified these organisms on the basis of a large number of biochemical reactions. These findings have been summarized in over 150 clearly labeled tables which in toto contain a wealth of information.

The first four chapters essentially lay the groundwork for the purpose of the book, the detailed description and identification of the Enterobacteriaceae. These chapters describe in sufficient detail the taxonomy, nomenclature, isolation and identification techniques, biochemical reactions, and antigens of these organisms. Each of the twelve genera are then detailed. The last chapter is a description of necessary laboratory media and reagents, presented in "cook-book" fashion which should be of considerable value to laboratory technicians.

From a physician's standpoint, this book would interest only those with a particular fascination for the Enterobacteriaceae. However, it is an indispensable item for every academically oriented bacteriology laboratory and would be a welcome addition to all laboratories concerned with bacterial cultures.

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obituaries

Dr. A. Magruder MacDonald, D.C. Coroner for 28 years, died October 5, 1972. He resided at 5115 Lowell Lane, N.W.

A native of Washington, D.C., he received his M.D. degree from Georgetown University and served his internship at the old Emergency Hospital.

He served in the Army Medical Corps in 1918 and was one of the American physicians loaned to the British forces in World War I.

Formerly deputy coroner of the District of Columbia, Dr. MacDonald was named Coroner in 1934 and served in that office until 1962. During his years as Coroner he testified at inquests in over 1,000 cases. He was said to have developed the relation between the police homicide squad and the coroner's office into one of the finest murder investigation systems in the country. As Coroner, he was the only law officer empowered to issue a subpoena to be served on the U.S marshal or to enter an embassy premise to investigate a violent death.

Dr. MacDonald lectured for several years on medical jurisprudence at Georgetown University and at the FBI's National Police Academy.

In 1965 he was elected President of Sibley Memorial Hospital's medical staff, where he had served for over 40 years. He was one of the original members of the medical council there. He had also served on the staff at Providence and Casualty hospitals.

He was a Life member and past President of the Medical Society of D.C., having served during the year 1942-1943. He had also been Secretary of the Maryland-D.C.-Virginia Medical Society.

Dr. MacDonald is survived by his wife, Helen, of the home; a daughter, Andrea Moran, of Potomac, Maryland, and three grandchildren.

Frank S. Ashburn, M.D.
1914–1972

Dr. Frank S. Ashburn, chief of thoracic surgery at Doctors Hospital and at Sibley Memorial Hospital, died October 14, 1972. He resided at 4911 Palisade Lane, N.W.

Born in Emory, Texas, he received his undergraduate and M.D. degrees from the University of Texas. He interned at Ancker Hospital in St. Paul, Minnesota and was a fellow in medicine there. He became a fellow at the Mayo Clinic in Rochester, Minnesota where he trained in both general and thoracic surgery. His association there was interrupted by his service in the U.S. Navy during World War II.

He served at the Naval Hospital here and in the Pacific theater with the first defense battalion of the Marine Corps from 1942 until 1947. During the invasion of the Marshall Islands, he introduced the first mobile surgery unit for amphibious operations for which he received the presidential unit citation.

Dr. Ashburn had been chief of thoracic surgery at Doctors Hospital since 1948 and at Sibley Memorial Hospital since 1958. He had also been associate professor of thoracic surgery at Georgetown University School of Medicine since 1948.

An Active member of the Medical Society of D.C., Dr. Ashburn made a great contribution, giving his time to many committees and Society activities. He also held membership in the Eastern Surgical Association, the American Association for Thoracic Surgery, and the Southern Thoracic Surgical Association. He published many articles on advancements in both general and thoracic surgery and his research led to the formulation of techniques basic to pulmonary surgery.

He is survived by his wife, Ida N., two sons, Frank Jr. and Thomas; and two daughters, Ann and Ida, all of the home; a married daughter, Mayfair Head of Lufkin, Texas, and a sister, Mrs. Dick P. Wood, of Dallas, Texas.

Richard H. Edenbaum, M.D.
1933–1972

Dr. Richard H. Edenbaum, a Maryland Internist, died October 17, 1972. He resided at 8205 Kenfield Court, Bethesda, Maryland.

Born in New York, Dr. Edenbaum received his undergraduate education at Columbia University and his M.D. degree from New York University College of Medicine. He interned at the Jewish Hospital of Brooklyn and served residencies there and at the Veterans Administration Hospital of Brooklyn.

He had practiced Internal Medicine in the Washington area for more than 10 years. He was on the staff of Georgetown and Holy Cross hospitals and the Washington Sanitarium and Hospital. He was also on the teaching staff of Georgetown University School of Medicine.

As Associate member of the Medical Society of D.C., he was also a member of the Maryland State Medical Association and the Society of Internal Medicine.

Dr. Edenbaum is survived by his wife, Marcia Rosencrantz Edenbaum; three children, David, Jonathan and Lisa, all of the home; and his parents, Mr. and Mrs. Edward M. Edenbaum, of New York City.

S. Percy Feldstein, M.D.
1902–1972

Dr. S. Percy Feldstein, who practiced Internal Medicine here and abroad for more than 35 years, died August 16, 1972 while vacationing in Bad Neuenahr, West Germany. He resided most recently in Israel and maintained a residence here at 3003 Van Ness Street, N.W.

Born in West Germany, he received his medical degree in Hamburg, West Germany, and served his internship and residency at Heine Hospital there. He came to Washington, D.C. in 1959, where he engaged in private practice at 6425 14th Street, N.W. He left to practice internal medicine in Kiron, Israel in 1964.

Dr. Feldstein was an Associate member of the Medical Society of D.C. He is survived by his wife, Marianne, of the home address.

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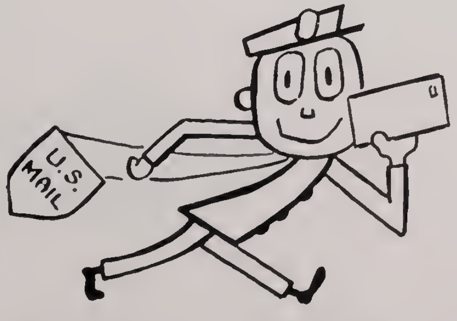

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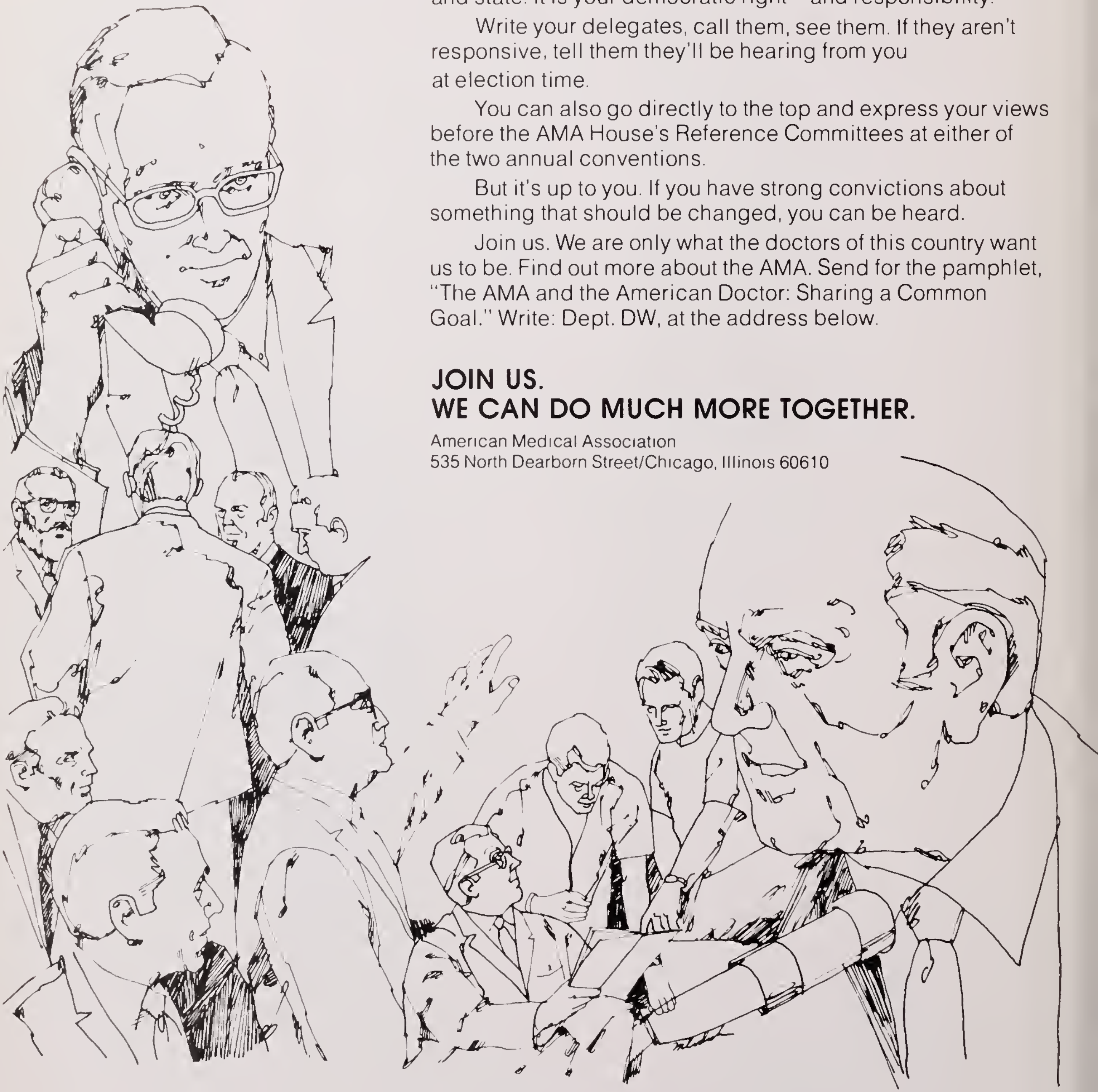

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